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# GROWTH OF MAJOR LONG BONES IN HEALTHY CHILDREN

## A PRELIMINARY REPORT ON SUCCESSIVE ROENTGENOGRAMS OF THE EXTREMITIES FROM EARLY INFANCY TO TWELVE YEARS OF AGE

MARION M. MARESH, M.D.

DENVER

A few years ago an associate and I presented a method for the study of segmental skeletal growth—the measurement of length of bones from roentgenograms.[1] These measurements were shown to be more reliable than anthropometric measurements in calculating the growth in length of the extremities of a group of 80 infants. This same method of measuring the length of the humerus, radius, ulna, femur, tibia and fibula from seriatim roentgenograms of the extremities during childhood has been utilized in the collection of data for the present study. The rates of growth of the individual long bones of the extremities, together with the relationships that exist between them and the rate of general skeletal growth, seem to add considerably to one's appreciation of the orderliness of the skeletal growth process. They add also to an understanding of the variations in bodily proportions which differentiate one child from another.

### MATERIAL

The 113 subjects for this study (52 girls and 61 boys) make up most of the group of average healthy children on whom the members of the staff of the Child Research Council have been collecting data for many years. While these children are being followed from birth to maturity many different types of investigation are being carried out. Several years ago we of this staff began to include roentgenograms of the left arm and leg at semiyearly intervals in this program, 671 sets of such films being now available for analysis. The number of children for whom roentgenograms of the long bones are being taken changes with the addition of each new baby to the study, and, of course, the number in each age range increases each year. At present, 18 of the children (16 per cent) have had less than a year's follow-up on roentgenograms of the long bones. There are 39 children (34.5 per cent) who have had roentgenograms of the long bones taken at intervals over a period of four years or longer, and 68 (60 per cent) have been followed for three years or longer. Represented in the 671 sets of roentgenograms are those of 34 children (30 per cent) who have had 8 to 11 sets taken since the beginning of this study, 63 children (56 per cent) who have had between 3 and 8 sets, and 16 children (14 per cent) who have had 1 or 2 sets each. This report does not include any roentgenograms on children over 12 years of age, since the number of older children is so few at this time that the data are insufficient for discussion. Limited studies are being carried out, however, on the children over 12 years of age, and it is probable that this material will accumulate in sufficient quantities to be of value in the next few years.

The present ages of the 113 children in this study are widely scattered between 4 months and 12 years. Thus the number of sets of roentgenograms in each age group, arranged in six month intervals, is also variable. However, there are 25 or more sets of individual data in each six month age group from 1½ through 10 years of age. As there are fewer subjects from 10½ to 12 years old, it is likely that the data for these age groups are not truly repre-

From the Child Research Council and the University of Colorado School of Medicine.

1. Maresh, M. M., and Deming, J.: The Growth of the Long Bones in Eighty Infants, Child Development **10**:91 (June) 1939.

sentative of the group as a whole. Growth is so rapid during the first year of life that smaller age intervals were used in collecting data for children in those age groups, and here again the numbers of subjects are too few to permit generalizations regarding the entire group. Further work on early infancy and the later preadolescent and adolescent years will be necessary before these data can be useful for cross-sectional analysis. The results of the work to date are, however, sufficiently interesting to warrant discussion of all the measurements made from the roentgenograms taken for children up to and including 12 years of age. These measurements are appended as table 5.

The roentgenographic technic has been kept as constant as is possible with children of such different ages. A 7½ foot (228.6 cm.) focus-film distance has been used throughout to reduce the distortion to a minimum. High speed screens have been used in the cassettes throughout this work, with the result that some of the finer details of bone structure have been sacrificed in the interest of speed. Infants are held in position with the arm or leg flat against the cassette in an anteroposterior position, while the older children stand on a platform in front of the cassette holder with the arm or leg in contact with the surface of the cassette. Exposures are made with 100 milliamperes and as short a time as is feasible. For infants, one twentieth of a second and 65 to 75 kilovolts have been found to be adequate exposure. For older children, time and voltage are varied from one tenth to nine twentieths of a second and from 60 to 75 kilovolts, depending on the size and musculature of the child. Except during early infancy, films are scheduled to be taken at the yearly and half-yearly visits to the Child Research Council.

The lengths of the major long bones of the arm and leg (humerus, radius, ulna, femur, tibia and fibula) are easily measured from the roentgenograms. These measurements are made in the simplest possible manner—the greatest possible length of the shaft, taken along the long axis of the bone from epiphysial line to epiphysial line, is measured to the nearest half-millimeter with a Lufkin steel ruler. In most instances this measurement of greatest length is from approximately the midpoint of one diaphysis to the midpoint of the other, but no attempt is made to determine that midpoint, since that introduces another set of variables and cannot be applied to roentgenograms on which slight rotation of a bone is obvious. All measurements have been made and checked by the same person (M. M.).

Since all the children in the Child Research Council study are measured by the anthropometrist at each yearly and half-yearly examination, it is possible to record their total heights for the same day on which the roentgenograms of the long bones are taken. In about 60 instances the heights were not taken by the staff anthropometrist but by other staff members with different technics. These 60 values were not recorded or utilized in this study.

## COMMENT

*Growth in Length of the Long Bones.*—Sex differences in the growth rates of boys and girls are well established in the literature, and it seems almost certain that these same differences are to be found in the growth in length of the long bones of the extremities. Stuart and Dwinell [2] have recently reported a significant sex difference in tibial lengths between 6 and 10 years of age. In spite of that fact, I have combined the data on boys and on girls for the present study, since the number of children in each age group is small and since my colleagues and I have been able to detect few sex differences in growth rates of bones up to 10 years of age in this group. Admitting the probability that minor differences are present, it seems likely that in so small a series some of the sex differences may be masked by the chance selection of subjects. We plan as data accumulate during the next few years to explore more thoroughly the difference in growth levels of each bone for boys and for girls. For the present, however, we are more concerned with the comparison of one child with the others in the group, regardless of sex.

Scatter charts showing the lengths of these long bones plotted against age represent the initial step in studying growth of bone. That growth in length of bone is extremely uniform is the first impression one gets from inspection of these charts. Length of the femur (the most rapidly growing of the six bones measured) and length of the radius (the slowest growing of these bones) are graphed against age in chart 1 *a* and *b*. In a few instances values for repeated measurements on the

2. Stuart, H. C., and Dwinell, P. H.: The Growth of Bone, Muscle and Overlying Tissues in Children Six to Ten Years of Age as Revealed by Studies of Roentgenograms of the Leg Area, Child Development **13**:195 (Sept.) 1942.

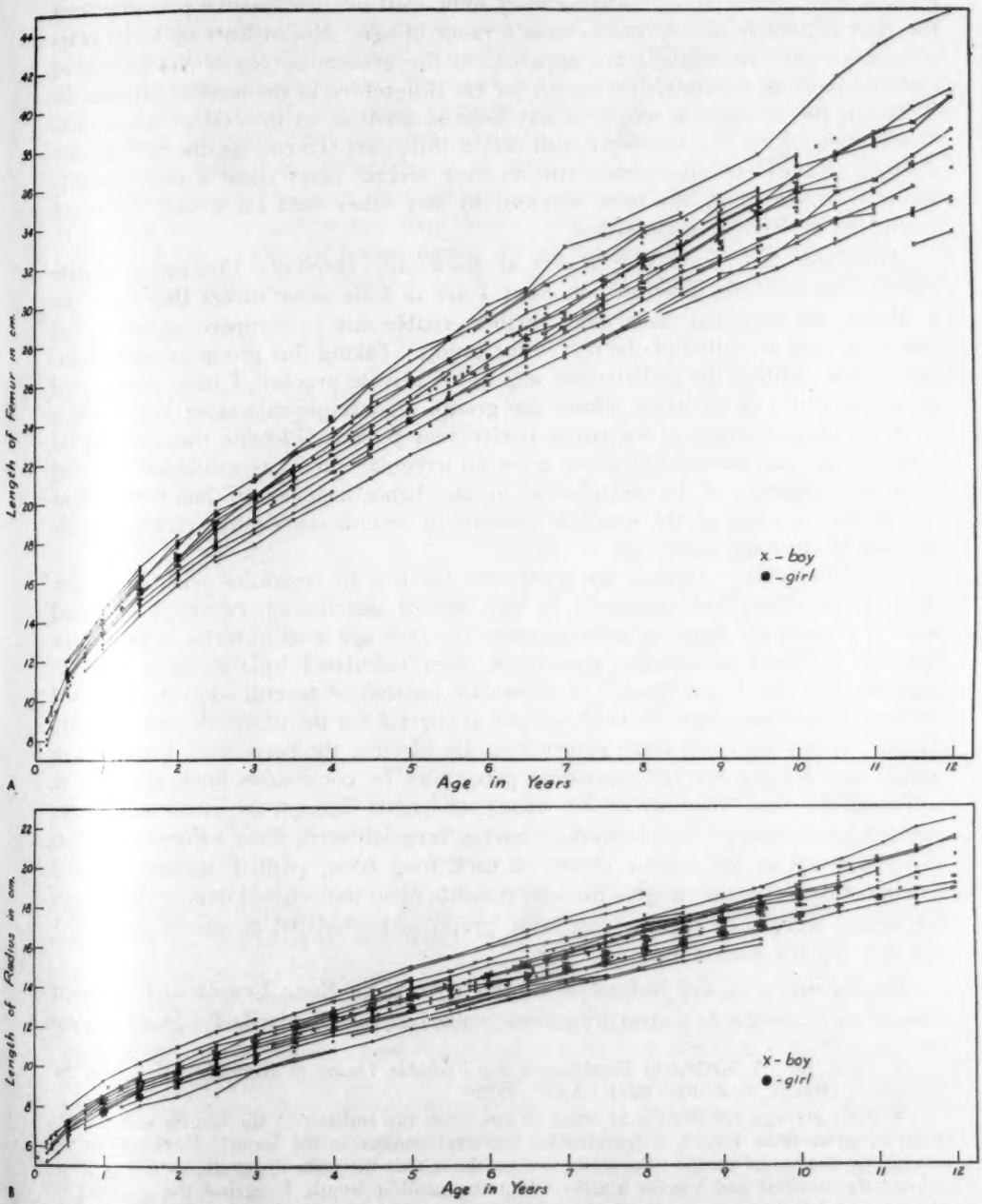



Chart 1.—Measurements of length of the femur and of the radius plotted against age in years. Successive measurements for the same child are connected by straight lines in illustrative cases. *A*, 670 measurements of length of the femur of 52 girls and 61 boys; *B*, 669 measurements of length of the radius of 52 girls and 61 boys.

same child are connected with straight lines to illustrate the regularity of the growth rate. One sees at once that each child tends to maintain his position within the group throughout the period followed. The variation in timing of the adolescent growth spurt in different children causes some shift in their relative position, since the start of adolescence covers so wide a range of age. But at least up to 10 years of age, no obvious changes are apparent in the growth curves of the individual children in these scatter charts except for the differences in the level of attainment. Although the increase in length of any bone is small in an interval of six months —less than 1.5 cm. for the femur and only a little over 0.5 cm. for the radius after 2 years of age—repeated measurements over several years show a more orderly increase in size than has been revealed by any other data on growth we have assembled or have seen published.

*Standards for Lengths of Bones at Each Age Interval.*—Obviously, scatter charts such as those illustrated in chart 1 are of little value unless they help one to define the expected range and, further, enable one to compare an individual child's growth with that of the rest of the group. Taking this group as representative of the children the pediatrician sees in his private practice, I have endeavored to set up zones of variation within the group. No single calculated curve seems to fit the data throughout the entire twelve year period. Plotting the arithmetical mean of each six month age group gives an irregular curve, as would be expected from the weighting of the mean values by the chance inclusion of data for some of the largest or some of the smallest children in several consecutive class intervals and not in later age groupings.

Since the values in each age group are too few to determine whether or not they can be described accurately by the normal distribution curve, it appeared wise to present the range of measurements for each age level in terms of percentile rankings. These percentile values have been calculated by the ogive method, suggested by Dr. Edith Boyd.[3] The method consists of several steps as described by her [4] some years ago. It is illustrated in chart 2 for the ninetieth, seventy-fifth, fiftieth, twenty-fifth and tenth percentiles. By plotting the percentiles for each age period and joining the corresponding percentiles by continuous lines, one is able to depict the zone distributions of values for length through the entire age range under consideration. This procedure having been followed, these values were then superimposed on the scatter charts of each long bone, plotted against age and smoothed by inspection to give the best possible fit to the original data. The values for length were then read off the large graph and tabulated as shown in table 1 (*A* and *B*) for each bone.

*Consideration of the Individual Child's Growth in Bone Length as Compared with Group Standards.*—Accepting these values as the standards for length of the

3. Boyd, E.: A Method of Establishing the Probable Limits of Normal Variation in the Weight of Organs, Anat. Rec. **62**:1 (April) 1935.

4. First arrange the lengths in order of size from the smallest to the largest and number them in series from 1 to n, n representing the total number in the series. Mark off on the y axis the number of cases in the series and on the x axis the units of length, extending them beyond the smallest and longest lengths. Plot the smallest length, 1, against the midpoint of the interval 0 to 1 on the y axis, the next length, 2, against the midpoint of the interval 1 to 2, and so forth, until the longest length is plotted against the midpoint of the last interval. Then draw in by inspection, either free hand or with a French curve, a smooth S-shaped curve through the central trend of the plotted points. One finds the number of cases expected at any desired percentile by computing that percentage of the total number of cases. Draw in a horizontal line for the percentile at its corresponding number on the y axis until it intersects the smooth curve. Then drop a perpendicular line from this point to the x axis and read the length for the given percentile from it.

long bones, one is able then to produce individual graphs for each child showing the status of the lengths of his bones as compared with the standards set up from the group data. No standards have been set up for lengths of the bones before 6 months of age, because of insufficient numbers of subjects. Therefore, most of the youngest children's growth curves cannot yet be used for comparison with the growth standards for the group. Omitting these youngest children as well as a few of the older children who have had fewer than three sets of measurements leaves 90 children—47 boys and 43 girls—whose bone growth could be studied further and analyzed. For the most part, graphs of their data bear out the impression gained from the scatter charts in chart 1: Each child tends to maintain his position in the group on successive measurements. Approximately half of the individual subjects follow the zone trends, with no significant shift in relative position of any of the six bones during the period when measurements have been made. For example, in chart 3 *A*, at 7 years the length of the humerus falls

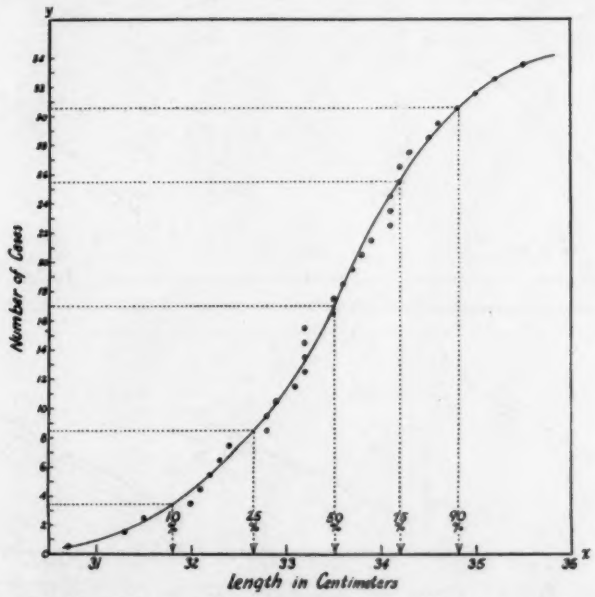



Chart 2.—Illustration of the method of obtaining values for length from the ogive for any desired percentile. Since one factor, in this series the age level, is a constant, ogives were drawn for each of the age groups for each bone.

between the tenth and the twenty-fifth percentile, and all succeeding measurements up to the last one, at 11 years, are in this same zone. The radius has remained consistently just below the twenty-fifth percentile, the ulna near the twenty-fifth percentile, the femur just below the tenth percentile and the tibia and the fibula between the twenty-fifth and the fiftieth percentile. In chart 3 (*A, B, C* and *D*) are sample charts of the group of 46 children (51 per cent), including 26 girls and 20 boys, whose relative positions have remained stable through successive measurements.

The other 44 children all show some shift in the relative lengths of one or more bones on their graphs. A second type of growth curve is seen in 14 children (15 per cent) in whom the increase or decrease in position has affected at least five of the six bones. This seems to indicate a general spurt or lag in growth over the rest of the group. Chart 4 *A* is illustrative of the few who show relative

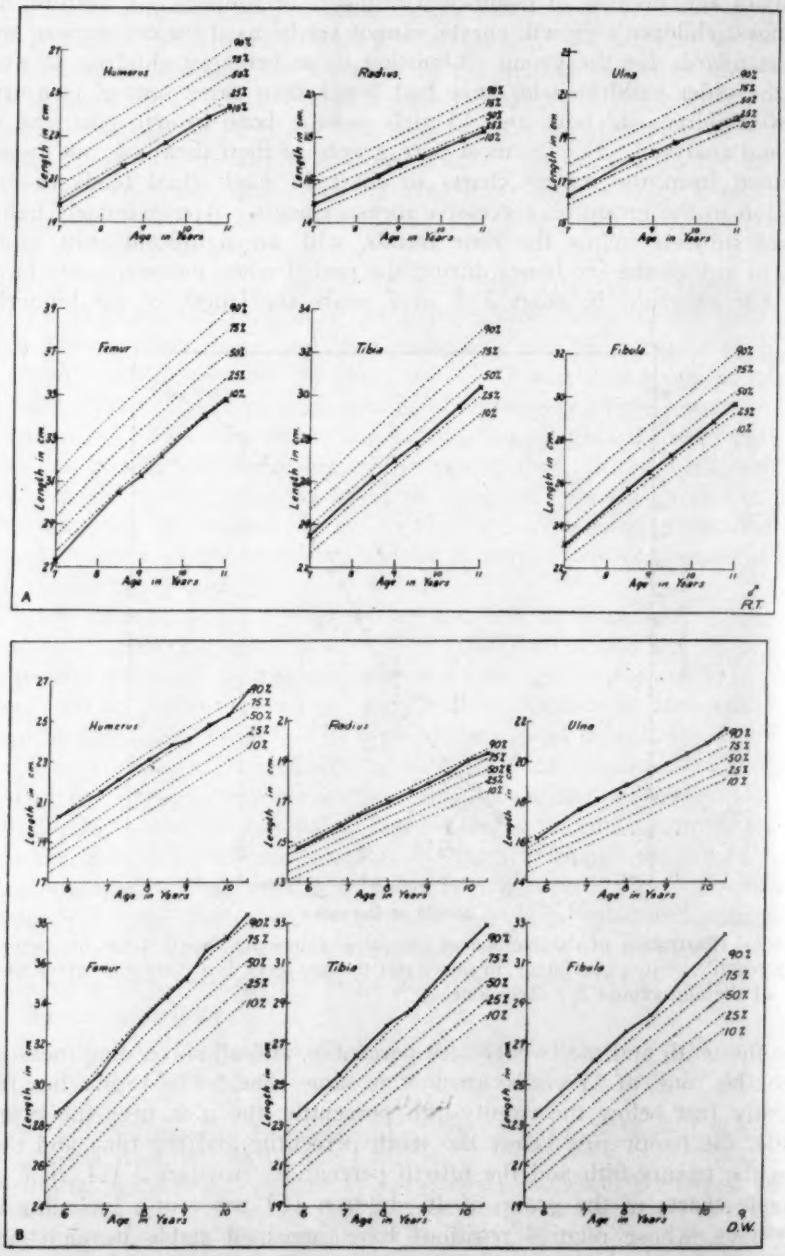
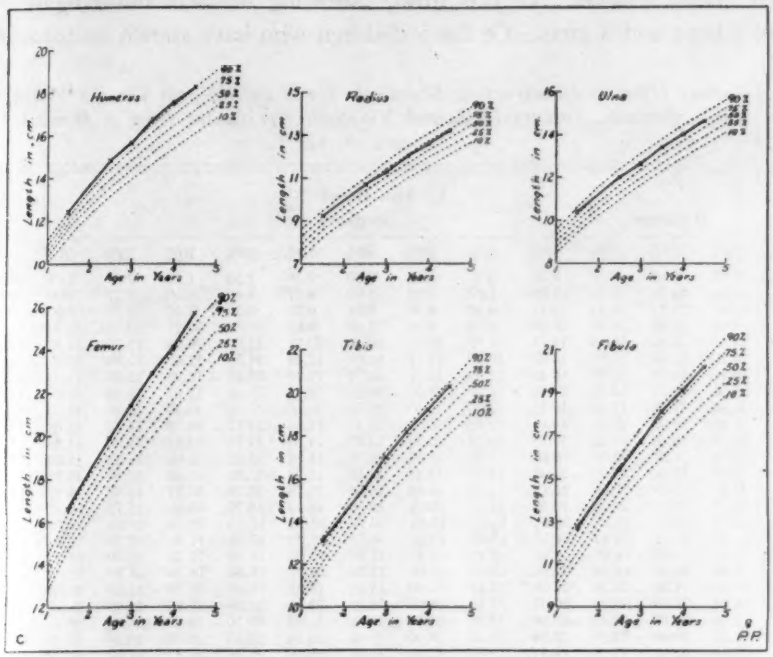



Chart 3.—Growth in length of six long bones for 4 of the 46 children whose rates of growth of the bones were not significantly different from the percentile trends used as standards. The percentile standards are given in table 1.



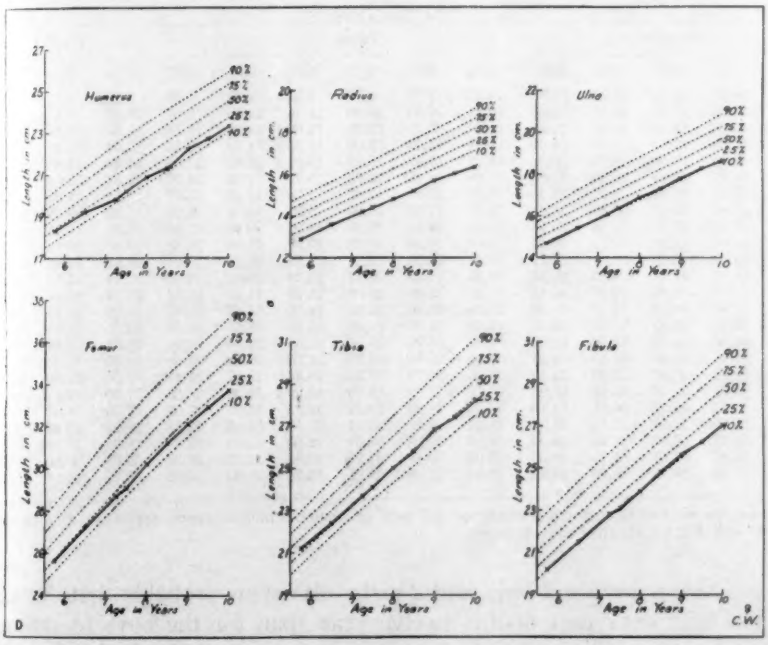




**Chart 3 continued.**

increase in length of all bones on succeeding measurements. Chart 4 *B* illustrates the curves for 1 of the 9 children (10 per cent) with gradual loss of position on successive measurements. In this group, showing uniform lowering of position, there are 6 boys and 3 girls. Of the 5 children who have shown uniform improve-

TABLE 1.—*Values Used in Constructing Standards for Bone Lengths for the Tenth, Twenty-Fifth, Fiftieth, Seventy-Fifth and Ninetieth Percentiles from 6 Months to 12 Years of Age* *

A. Arm Bones

| Age, Yr. | Humerus | | | | | Radius | | | | | Ulna | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 10% | 25% | 50% | 75% | 90% | 10% | 25% | 50% | 75% | 90% | 10% | 25% | 50% | 75% | 90% |
| ½ | 8.35 | 8.65 | 8.96 | 9.26 | 9.53 | 6.13 | 6.58 | 6.90 | 7.18 | 7.50 | 7.19 | 7.47 | 7.78 | 8.06 | 8.31 |
| 1 | 10.04 | 10.34 | 10.60 | 10.84 | 11.09 | 7.27 | 7.62 | 7.92 | 8.27 | 8.60 | 8.50 | 8.77 | 9.00 | 9.30 | 9.60 |
| 1½ | 11.23 | 11.62 | 11.91 | 12.19 | 12.47 | 8.22 | 8.60 | 8.94 | 9.21 | 9.50 | 9.46 | 9.72 | 10.00 | 10.33 | 10.63 |
| 2 | 12.29 | 12.64 | 13.00 | 13.38 | 13.68 | 8.96 | 9.36 | 9.68 | 9.96 | 10.28 | 10.27 | 10.57 | 10.88 | 11.20 | 11.59 |
| 2½ | 13.18 | 13.55 | 13.93 | 14.39 | 14.74 | 9.65 | 10.02 | 10.40 | 10.71 | 11.00 | 10.95 | 11.25 | 11.62 | 12.00 | 12.38 |
| 3 | 13.92 | 14.39 | 14.80 | 15.28 | 15.68 | 10.30 | 10.74 | 11.09 | 11.44 | 11.80 | 11.60 | 11.98 | 12.37 | 12.72 | 13.11 |
| 3½ | 14.65 | 15.20 | 15.70 | 16.20 | 16.58 | 11.01 | 11.37 | 11.70 | 12.12 | 12.48 | 12.29 | 12.65 | 13.08 | 13.38 | 13.78 |
| 4 | 15.40 | 15.98 | 16.48 | 16.99 | 17.40 | 11.53 | 12.00 | 12.33 | 12.73 | 13.10 | 12.91 | 13.28 | 13.69 | 14.02 | 14.42 |
| 4½ | 16.10 | 16.68 | 17.20 | 17.73 | 18.23 | 12.10 | 12.50 | 12.90 | 13.30 | 13.72 | 13.47 | 13.89 | 14.31 | 14.60 | 14.99 |
| 5 | 16.80 | 17.38 | 17.90 | 18.55 | 19.01 | 12.62 | 13.07 | 13.47 | 13.86 | 14.27 | 14.03 | 14.45 | 14.88 | 15.19 | 15.58 |
| 5½ | 17.51 | 18.07 | 18.59 | 19.35 | 19.88 | 13.11 | 13.54 | 13.92 | 14.38 | 14.71 | 14.51 | 14.93 | 15.38 | 15.69 | 16.12 |
| 6 | 18.19 | 18.78 | 19.31 | 20.05 | 20.63 | 13.54 | 13.98 | 14.38 | 14.82 | 15.21 | 15.00 | 15.41 | 15.86 | 16.24 | 16.68 |
| 6½ | 18.79 | 19.40 | 19.96 | 20.76 | 21.40 | 14.00 | 14.42 | 14.83 | 15.28 | 15.67 | 15.42 | 15.84 | 16.35 | 16.80 | 17.25 |
| 7 | 19.38 | 20.01 | 20.68 | 21.48 | 22.11 | 14.48 | 14.87 | 15.30 | 15.78 | 16.19 | 15.87 | 16.28 | 16.82 | 17.30 | 17.78 |
| 7½ | 19.98 | 20.62 | 21.31 | 22.14 | 22.83 | 14.92 | 15.31 | 15.80 | 16.24 | 16.70 | 16.32 | 16.78 | 17.27 | 17.80 | 18.33 |
| 8 | 20.58 | 21.20 | 21.96 | 22.78 | 23.52 | 15.35 | 15.81 | 16.27 | 16.70 | 17.18 | 16.79 | 17.25 | 17.78 | 18.28 | 18.80 |
| 8½ | 21.19 | 21.83 | 22.61 | 23.42 | 24.10 | 15.83 | 16.25 | 16.72 | 17.20 | 17.65 | 17.28 | 17.70 | 18.26 | 18.79 | 19.33 |
| 9 | 21.80 | 22.46 | 23.26 | 24.07 | 24.72 | 16.29 | 16.73 | 17.20 | 17.67 | 18.10 | 17.75 | 18.20 | 18.72 | 19.26 | 19.81 |
| 9½ | 22.40 | 23.09 | 23.90 | 24.68 | 25.31 | 16.70 | 17.20 | 17.70 | 18.17 | 18.62 | 18.23 | 18.70 | 19.21 | 19.80 | 20.38 |
| 10 | 23.05 | 23.75 | 24.51 | 25.28 | 25.90 | 17.10 | 17.60 | 18.10 | 18.67 | 19.15 | 18.68 | 19.15 | 19.65 | 20.27 | 20.88 |
| 10½ | 23.67 | 24.40 | 25.20 | 25.91 | 26.55 | 17.52 | 18.05 | 18.59 | 19.14 | 19.58 | 19.13 | 19.59 | 20.18 | 20.80 | 21.40 |
| 11 | 24.20 | 24.98 | 25.80 | 26.49 | 27.20 | 18.00 | 18.50 | 19.00 | 19.62 | 20.10 | 19.60 | 20.09 | 20.69 | 21.30 | 21.90 |
| 11½ | 24.80 | 25.59 | 26.40 | 27.09 | 27.78 | 18.45 | 18.90 | 19.46 | 20.14 | 20.69 | 20.08 | 20.58 | 21.20 | 21.82 | 22.45 |
| 12 | 25.39 | 26.19 | 27.01 | 27.70 | 28.41 | 18.80 | 19.30 | 19.88 | 20.58 | 21.15 | 20.47 | 21.01 | 21.70 | 22.38 | 23.00 |

B. Leg Bones

| | Femur | | | | | Tibia | | | | | Fibula | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | 10% | 25% | 50% | 75% | 90% | 10% | 25% | 50% | 75% | 90% | 10% | 25% | 50% | 75% | 90% |
| ½ | 10.34 | 10.77 | 11.24 | 11.56 | 11.88 | 8.46 | 8.78 | 9.00 | 9.35 | 9.73 | 8.00 | 8.28 | 8.57 | 8.92 | 9.23 |
| 1 | 12.80 | 13.15 | 13.51 | 13.90 | 14.32 | 10.31 | 10.60 | 10.90 | 11.26 | 11.66 | 9.74 | 10.10 | 10.48 | 10.90 | 11.30 |
| 1½ | 14.76 | 15.08 | 15.50 | 15.97 | 16.42 | 11.73 | 12.15 | 12.50 | 12.90 | 13.31 | 11.18 | 11.60 | 12.12 | 12.55 | 13.05 |
| 2 | 16.13 | 16.61 | 17.14 | 17.60 | 18.04 | 13.07 | 13.48 | 13.96 | 14.35 | 14.72 | 12.63 | 13.08 | 13.62 | 14.15 | 14.62 |
| 2½ | 17.50 | 18.00 | 18.50 | 19.09 | 19.52 | 14.22 | 14.63 | 15.13 | 15.57 | 16.05 | 13.90 | 14.40 | 15.02 | 15.58 | 16.00 |
| 3 | 18.74 | 19.31 | 19.84 | 20.52 | 21.00 | 15.18 | 15.68 | 16.29 | 16.77 | 17.26 | 14.97 | 15.57 | 16.18 | 16.80 | 17.33 |
| 3½ | 19.96 | 20.53 | 21.24 | 21.94 | 22.50 | 16.16 | 16.68 | 17.37 | 17.94 | 18.40 | 15.93 | 16.54 | 17.20 | 17.88 | 18.43 |
| 4 | 21.19 | 21.80 | 22.48 | 23.28 | 23.95 | 17.07 | 17.63 | 18.36 | 18.97 | 19.50 | 16.93 | 17.58 | 18.18 | 18.86 | 19.41 |
| 4½ | 22.30 | 22.98 | 23.73 | 24.58 | 25.18 | 18.05 | 18.61 | 19.30 | 19.97 | 20.48 | 17.86 | 18.48 | 19.10 | 19.83 | 20.50 |
| 5 | 23.50 | 24.08 | 24.82 | 25.79 | 26.43 | 19.00 | 19.58 | 20.32 | 20.97 | 21.48 | 18.81 | 19.43 | 20.08 | 20.81 | 21.49 |
| 5½ | 24.62 | 25.20 | 26.00 | 26.92 | 27.71 | 19.98 | 20.58 | 21.23 | 21.92 | 22.49 | 19.72 | 20.34 | 21.03 | 21.76 | 22.43 |
| 6 | 25.64 | 26.31 | 27.14 | 28.02 | 28.88 | 20.89 | 21.42 | 22.11 | 22.78 | 23.45 | 20.50 | 21.24 | 21.93 | 22.70 | 23.40 |
| 6½ | 26.70 | 27.37 | 28.25 | 29.25 | 30.10 | 21.80 | 22.30 | 23.00 | 23.72 | 24.43 | 21.52 | 22.16 | 22.84 | 23.63 | 24.38 |
| 7 | 27.71 | 28.40 | 29.32 | 30.33 | 31.30 | 22.68 | 23.20 | 23.93 | 24.70 | 25.47 | 22.37 | 22.99 | 23.70 | 24.52 | 25.27 |
| 7½ | 28.70 | 29.45 | 30.41 | 31.50 | 32.46 | 23.48 | 24.10 | 24.82 | 25.59 | 26.48 | 23.12 | 23.78 | 24.58 | 25.42 | 26.17 |
| 8 | 29.69 | 30.50 | 31.51 | 32.50 | 33.50 | 24.29 | 24.98 | 25.77 | 26.55 | 27.50 | 23.93 | 24.62 | 25.43 | 26.33 | 27.05 |
| 8½ | 30.67 | 31.48 | 32.50 | 33.50 | 34.44 | 25.12 | 25.88 | 26.70 | 27.50 | 28.40 | 24.69 | 25.40 | 26.31 | 27.18 | 27.89 |
| 9 | 31.50 | 32.41 | 33.40 | 34.50 | 35.32 | 25.98 | 26.70 | 27.55 | 28.44 | 29.37 | 25.48 | 26.20 | 27.06 | 27.98 | 28.68 |
| 9½ | 32.40 | 33.22 | 34.36 | 35.42 | 36.25 | 26.75 | 27.52 | 28.39 | 29.29 | 30.28 | 26.25 | 27.00 | 27.87 | 28.78 | 29.50 |
| 10 | 33.30 | 34.17 | 35.20 | 36.31 | 37.10 | 27.50 | 28.33 | 29.21 | 30.25 | 31.17 | 27.08 | 27.79 | 28.68 | 29.65 | 30.39 |
| 10½ | 34.12 | 35.00 | 36.06 | 37.18 | 38.00 | 28.38 | 29.20 | 30.12 | 31.18 | 32.15 | 27.78 | 28.55 | 29.46 | 30.45 | 31.23 |
| 11 | 34.90 | 35.72 | 36.82 | 37.99 | 38.90 | 29.18 | 29.97 | 30.92 | 32.06 | 33.02 | 28.48 | 29.25 | 30.19 | 31.23 | 32.05 |
| 11½ | 35.68 | 36.47 | 37.61 | 38.71 | 39.69 | 30.02 | 30.80 | 31.77 | 32.88 | 33.97 | 29.22 | 30.03 | 30.98 | 32.01 | 32.85 |
| 12 | 36.40 | 37.20 | 38.36 | 39.50 | 40.49 | 30.86 | 31.66 | 32.63 | 33.78 | 34.82 | 29.88 | 30.72 | 31.77 | 32.79 | 33.67 |

* These values were obtained from study of 663 sets of measurements from seriatum roentgenograms of the left arm and leg on 51 girls and 60 boys.

ment in position there are 3 boys and 2 girls. It seems probable that the standards may be too high over part of this twelve year span for the boys to maintain and that a separation of the original data into separate standards for boys and for girls would show that there are fewer boys whose growth rate has been slower than that of the group standards. Another possibility to be considered is suggested by the fact that 5 of the 9 children who show relative loss of position were 4 years of age or less when the last measurements were made. Perhaps the stability

of the pattern of body proportions and relative lengths is not well established during early childhood and, as the spurt of growth associated with infancy slows down, either hereditary or environmental factors influencing growth become more

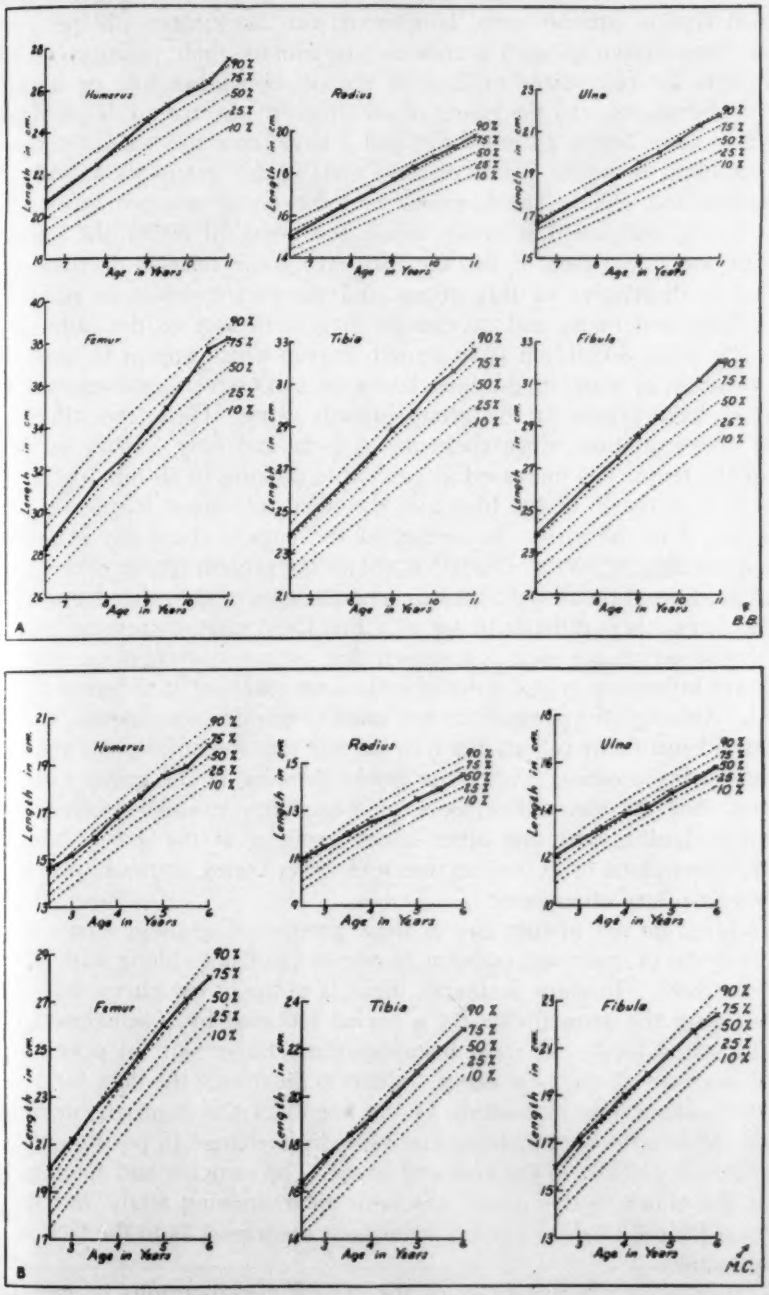


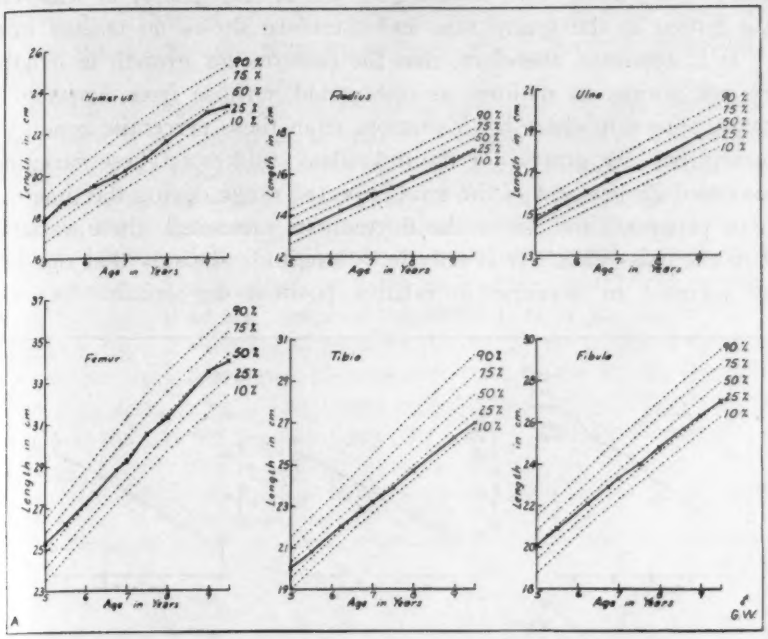

Chart 4.—*A*, growth in length of the bones for 1 of the 5 children whose rates of growth for at least five of the six bones were greater than the percentile zone trends used as standards. Successive measurements show an increase in percentile ranking during the period of study. *B*, growth in length of the bones for 1 of the 9 children whose rates of growth for at least five of the six bones were slower than the percentile zone trends used as standards. Successive measurements show a decrease in percentile ranking during the period of study.

dominant. It is possible that this group, who show a relative loss of position, would be larger if we had been able to follow all the 90 children from early infancy. Perhaps a clue as to the age period when growth rates become relatively stable may be found by continued observations on this group of younger children.

A third type of growth curve is observed for 24 children (27 per cent), who appear to have grown at such a rate as to maintain their position on successive measurements for two, three or four of the six bones but lose or improve their position in the others. In the group of 16 children who have relative decreases in the length of some bones, all but 1 girl and 2 boys show this loss in position of the tibia or the fibula or both. Three of the 4 girls in this group show loss in position of the radius and ulna. The humerus and the femur are not infrequently also noted as losing position, but never unless the bones of either the lower part of the arm or the lower part of the leg also have some relative decrease in length. Chart 5 *A* is illustrative of this group and shows some loss in position of the humerus, tibia and fibula and no change in growth rate of the radius, ulna and femur. The other 8 children have growth curves which appear to show improvement in position of some of the long bones on successive measurements while the other bones have grown at the group growth rates. Here, too, there seems to be a sex differentiation, since there are 6 girls and only 2 boys in this group. Length of the femur has increased in percentile ranking in all but 1 of the 8, of the humerus in 5 of the 8, of the ulna and the tibia next most frequently and of the fibula in only 1 of the girls. In neither of the boys is there any improvement of position in the tibia or fibula. Chart 5 *B* shows the growth of one of this group with improved position of the ulna and femur while lengths of the other bones parallel the zone trend lines. It is difficult to see why this third type of seemingly inconsistent growth should occur, unless it is assumed that certain unrecognized environmental or hereditary influences or sex differences become manifest at different times during childhood. Although the group is far too small to warrant conclusions, it would seem that one could justifiably call attention to the fact that the boys' tibias and fibulas are more often losing position, with other bones growing at the group rate, than any other bone, that the girls show more of a tendency toward lowered position of the radius and ulna, with the other bones growing at the group rate, and that the femur, either alone or in conjunction with other bones, shows improved position more often than any other bone.

Six children do not fit into any of these groups. For these 4 boys and 2 girls there is evidence of improved position of one or two bones along with some loss of position of others. In some instances there is a dip in the curve, which suggests some slowing of the growth rate for a period followed by a subsequent rise to or beyond the initial level. In most instances the relative shift in position does not exceed 25 per cent of the total range. Chart 6 illustrates the data for 1 of these 6 children with slight rise in position of the length of the humerus, definite rise in position of the lengths of the radius and ulna, little change in position of the femur and slight loss in position of the tibia and fibula. The complex and apparently erratic growth of the bones of this group has been an interesting study, but the number of subjects is far too small to justify any serious theorizing as to the factors involved in its production.

Thus, four main classifications of the 90 individual graphs of growth of the long bones seem permissible: a first group (51 per cent), with no apparent change in relative position of any of the bones on successive measurements, as shown in chart 3; a second group (15 per cent), with uniform lowering or increase in relative position on successive measurements, as shown in chart 4; a third group (27 per cent), with lowering or improvement in position of one, two, three or



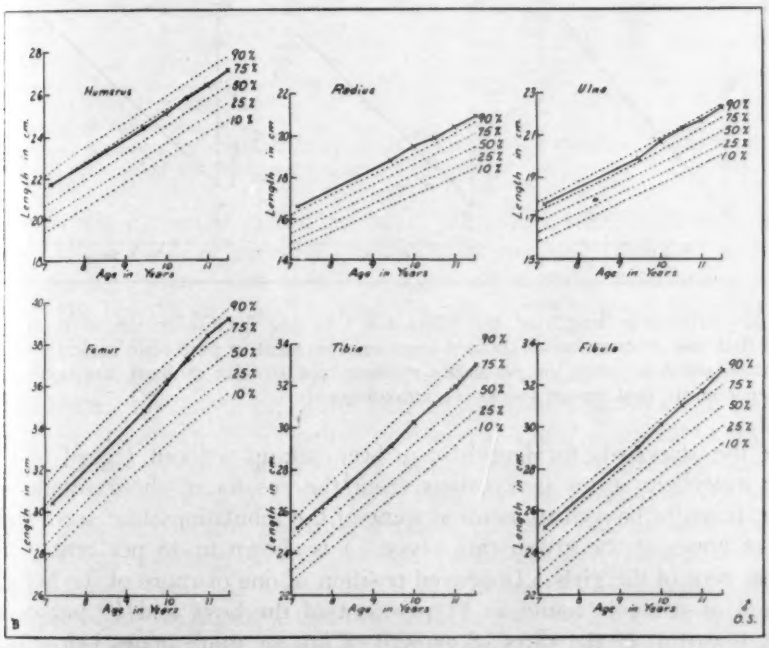




Chart 5.—*A*, growth in length of the bones for 1 of the 16 children whose rates of growth for two, three or four of the six bones were slower than the percentile zone trends used as standards while the growth of the other bones paralleled the percentile trends. *B*, growth in length of the bones for 1 of the 8 children whose rates of growth for two, three or four of the six bones were greater than the percentile zone trends used as standards while the growth of the other bones paralleled the percentile trends.

four bones and with no changes in relative position of the others, as illustrated in chart 5, and a fourth, smaller, group (7 per cent) with improved position of one or more bones along with lowering of others and usually at least one bone which has grown at the group rate and therefore shows no change in relative position. It is apparent, therefore, that the patterns for growth in length of the bones are not always so uniform as one would surmise from inspection of the scatter charts shown in chart 1. Variations from these percentile zone trends are seen in nearly half the graphs for the individual children. These variations have seldom exceeded 25 per cent of the total expected range during the time this work has been in progress, and, since the fluctuations are small, these variations are obscured in the mass data. It is only by a longitudinal study that one is able to detect the increase or decrease in relative position on seriatim measurements.

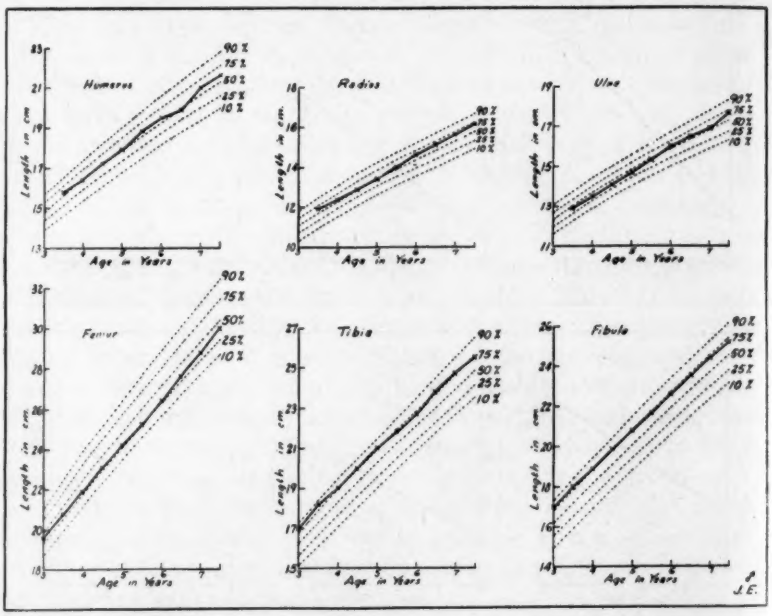



Chart 6.—Growth in length of the bones for 1 of the 6 children whose rates of growth were such that one or more bones showed some improvement in percentile ranking, one or more bones showed some lowering in percentile ranking and usually at least one bone showed no change in percentile ranking on successive measurements.

Since the standards for length have been set up without regard to sex, it is probably unwise to draw conclusions from the results of these simple analyses. However, it might be well to mention some of the tabulations that seem significant. Growth of bones at the group rate (type 1) is shown in 43 per cent of the boys and 60 per cent of the girls. Improved position of one or more of the bones during this period of study is found in 11 per cent of the boys and 19 per cent of the girls. A lowering of the rates of growth of one or more bones below the group standards is seen in 38 per cent of the boys and 16 per cent of the girls. The unclassified group (type 4) includes 8 per cent of the boys and 5 per cent of the girls. This seems to bear out the supposition that the standards are too high for the boys over at least part of the twelve year period.

When the 15 children ranking highest and the 15 ranking lowest for both stature and length of the long bones were chosen from the 90 children, there were 10 girls and 5 boys in each group. This suggests either that the range of variation is greater in girls than in boys between the ages of 1 and 10 years or that the data are not suitable for the determination of sex differences in growth rates at this time.

On the basis of relative lengths of bones as judged by the percentile rank of each bone, there is a suggestion of marked sex difference in the proportions of the arm and the leg. The humerus is relatively longer (i. e., has a higher percentile ranking) than the radius and ulna in twice as many girls as boys. It is relatively shorter (has a lower percentile ranking) than the radius and ulna in more than twice as many boys as girls. The humerus is relatively equal in rank to the radius and ulna in about equal numbers of boys and girls. The bones of the forearm are relatively longer than the bones of the lower part of the leg in four times as many boys as girls, but they are of equal rank with or the bones of the forearm are relatively shorter than the bones of the lower part of the leg in approximately a third more girls than boys. The bones which are relatively the longer are arm bones in more than twice as many boys as girls. The leg bones are relatively

TABLE 2.—*Coefficients of Correlation Between Lengths of Bones as Measured from the Roentgenograms of 52 Girls and 61 Boys*

| x | y | n | r | $\sigma_{y.x}$ | k |
|---|---|---|---|---|---|
| Humerus | Radius | 668 | 0.9941 | 0.40 | 0.73 |
| Humerus | Ulna | 668 | 0.9944 | 0.41 | 0.77 |
| Ulna | Radius | 669 | 0.9983 | 0.21 | 0.94 |
| Femur | Humerus | 667 | 0.9942 | 0.54 | 0.63 |
| Femur | Radius | 668 | 0.9915 | 0.48 | 0.46 |
| Femur | Tibia | 668 | 0.9951 | 0.65 | 0.83 |
| Femur | Fibula | 668 | 0.9949 | 0.65 | 0.81 |
| Tibia | Fibula | 669 | 0.9990 | 0.30 | 1.00 |

n = number of pairs of measurements.
r = coefficient of correlation.
$\sigma_{y.x}$ = vertical deviation (in centimeters) of observed values about calculated straight line.
k = slope (in centimeters per centimeter) of calculated straight line.

the longer in slightly more girls than boys. It would seem, therefore, that boys and girls are apt to be differently proportioned in these respects: (1) ratio of arm bones to leg bones, with the boys' arm bones being relatively longer, and (2) ratio of the bone of the upper part of the arm to the bones of the forearm, with the boys' forearms being relatively longer.

*Correlations Between the Length of One Bone and That of Another.*—Since the growth curves for length of all the long bones when charted against age follow the same general pattern, it seemed probable that there would be a high degree of correlation between the lengths of any two of these six major long bones. This has been found to be the case, as shown in table 2. The correlation coefficients are so high as to make it seem clear that a close relation exists between the lengths of the various bones during growth through the first twelve years of life.[5] Scatter charts illustrate this well, as shown in chart 7 *A* and *B*. The femur and radius, with the most widely divergent growth rates of the six long bones, have a correlation coefficient of 0.9915, and the tibia and fibula, which have nearly identical growth rates, have a correlation coefficient of 0.9990. That an essentially straight line rela-

5. When the data are separated into limited age intervals (e. g. two years ± two weeks) coefficients of correlation between the tibia and fibula for sample ages are as follows: 2 years ($n = 25$) $r = 0.9294$; 4 years ($n = 20$) $r = 0.9749$; 6 years ($n = 18$) $r = 0.9775$; 8 years ($n = 27$) $r = 0.9765$; 10 years ($n = 28$) $r = 0.9766$; 12 years ($n = 10$) $r = 0.9844$.

tionship exists between the lengths of any two of these six bones, as illustrated in chart 7 *A* and *B* for the first twelve years, suggest that such a relationship may also be present throughout adolescent and adult life. Adequate proof of this is lacking, but it seems probable that further work may well substantiate the thesis. This close relationship between the lengths of the bones of the extremities through-

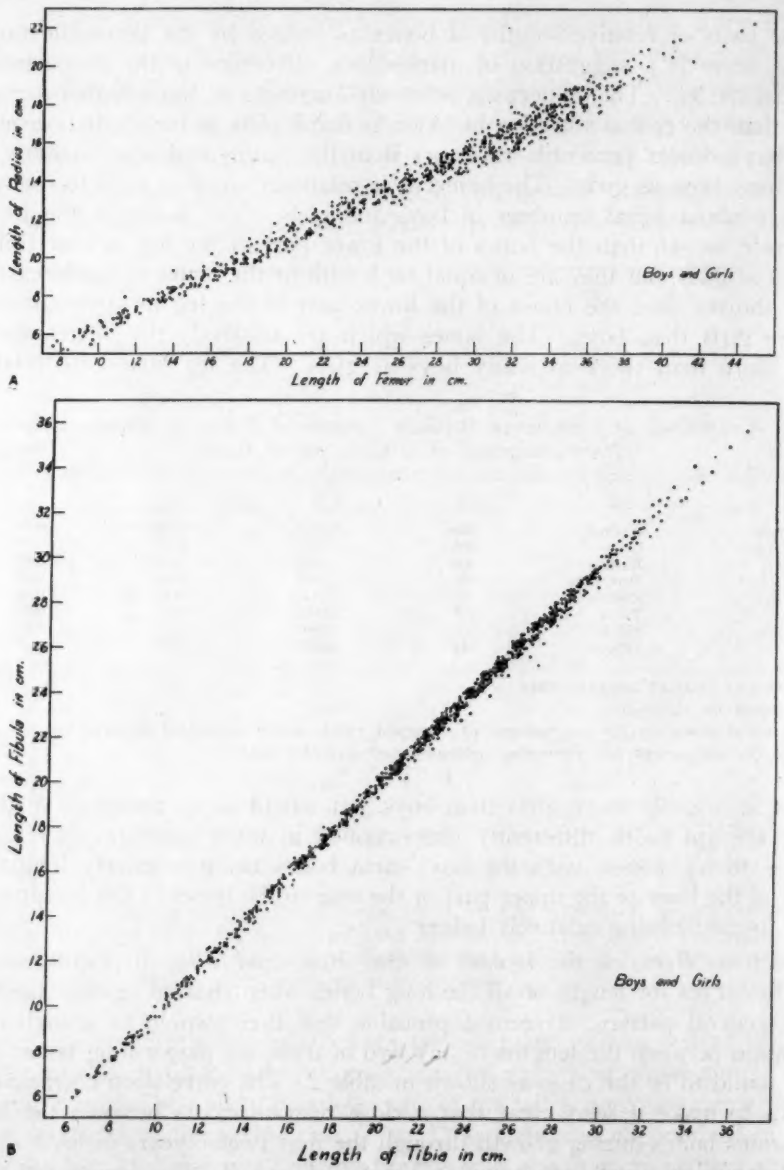



Chart 7.—Measurements of length of one bone plotted against those for length of another. The measurements were made from roentgenograms of 52 girls and 61 boys up to and including 12 years of age. *A*, 668 pairs of measurements of length of the femur and length of the radius plotted against each other; *B*, 669 pairs of measurements of length of the tibia and length of the fibula plotted against each other.

out the first twelve years of life demonstrates the regularity of skeletal growth and seems to warrant investigation of the relationship of length of these bones to the total body height.

*Correlation Between Length of the Long Bones and Stature.*—Measurements of the length of the infants are taken while they are in the supine position, while older children are measured erect. These measurements will be referred to collectively as "stature" henceforth, since they are partially measurements of length and partially measurements of height. Calculated correlation coefficients between lengths of the bones and stature are given in table 3 and range from 0.9913 to 0.9952. These coefficients are so high that one would expect an approximately straight line relationship between lengths of the long bones and stature, similar to that described for the bone to bone relation. Scatter charts of these measurements, however, do not bear this out, as seen in chart 8 *A* and *B*. Although such a straight line association is seen in the older children, there is a period during the early months of infancy when the lengths of the bones are proportionately greater in relation to stature than they are at later ages. After the first six months, although stature and lengths of the bones increase at different rates, these rates of increase bear a constant relationship to each other, the long bones growing steadily faster than the trunk. The implication of such a statement is that there must be a period of a few months after birth when the trunk is increasing in length at a greater rate than the rest of the axial skeleton. That this is probably true is

TABLE 3.—*Coefficients of Correlation Between Total Body Stature, Measured as Total Length or Total Height, and Lengths of the Six Major Long Bones of the Arm and Leg Measured from the Roentgenograms of 52 Girls and 61 Boys*

| x | y | n | r | $\sigma_{y.x}$ | k |
|---|---|---|---|---|---|
| Stature | Humerus | 610 | 0.9941 | 0.57 | 0.21 |
| Stature | Radius | 611 | 0.9913 | 0.50 | 0.15 |
| Stature | Ulna | 611 | 0.9916 | 0.52 | 0.16 |
| Stature | Femur | 611 | 0.9952 | 0.79 | 0.33 |
| Stature | Tibia | 612 | 0.9932 | 0.79 | 0.27 |
| Stature | Fibula | 612 | 0.9936 | 0.77 | 0.27 |

n = number of pairs of measurements.
r = coefficient of correlation.
$\sigma_{y.x}$ = vertical deviation (in centimeters) of observed values about calculated straight line.
k = slope (in centimeters per centimeter) of calculated straight line.

supported by Meredith and Knott's [6] study on the skelic index. They found that the skelic index $\frac{\text{length of the lower extremity} \times 100}{\text{stem length (vertex-rump, or sitting length, height)}}$ rises from close to zero at 4 weeks of embryonic life through 35 at 3 fetal months to 48 at 6 fetal months. Between 6 fetal months and 3 or 4 postnatal months the trend is inconclusive, but the authors suggested that the skelic index either remains almost constant over that age period or rises to about 52 at birth followed by a fall to about 49 at 3 postnatal months. From 3 postnatal months, the skelic index gradually increases to 93 at 13½ years of age. Thus, the shifting relationship between lengths of the bone and stature in early postnatal life can be explained in terms of the more rapidly growing stem length during these first few months. It seems probable from Meredith and Knott's report of a drop in skelic index after 13½ years that lengths of the bones throughout adolescence will not continue to show so nearly a straight line correlation with stature. Further work will be necessary to clarify that point.

*Ratios Between Bone Lengths and Stature and Between the Bones Themselves.*—The fact that an essentially straight line relationship exists after 6 months of age when one charts the length of any of these long bones against stature does not

6. Meredith, H. V., and Knott, V. B.: Changes in Body Proportions During Infancy and the Preschool Years: III. The Skelic Index, Child Development **9**:49 (March) 1938.

mean that the ratio between them is an unchanging one up to 12 years of age. For example, the length of the femur at 6 months of age is approximately 17 per cent of the stature for that age, with values ranging from 16.35 per cent to 18.81

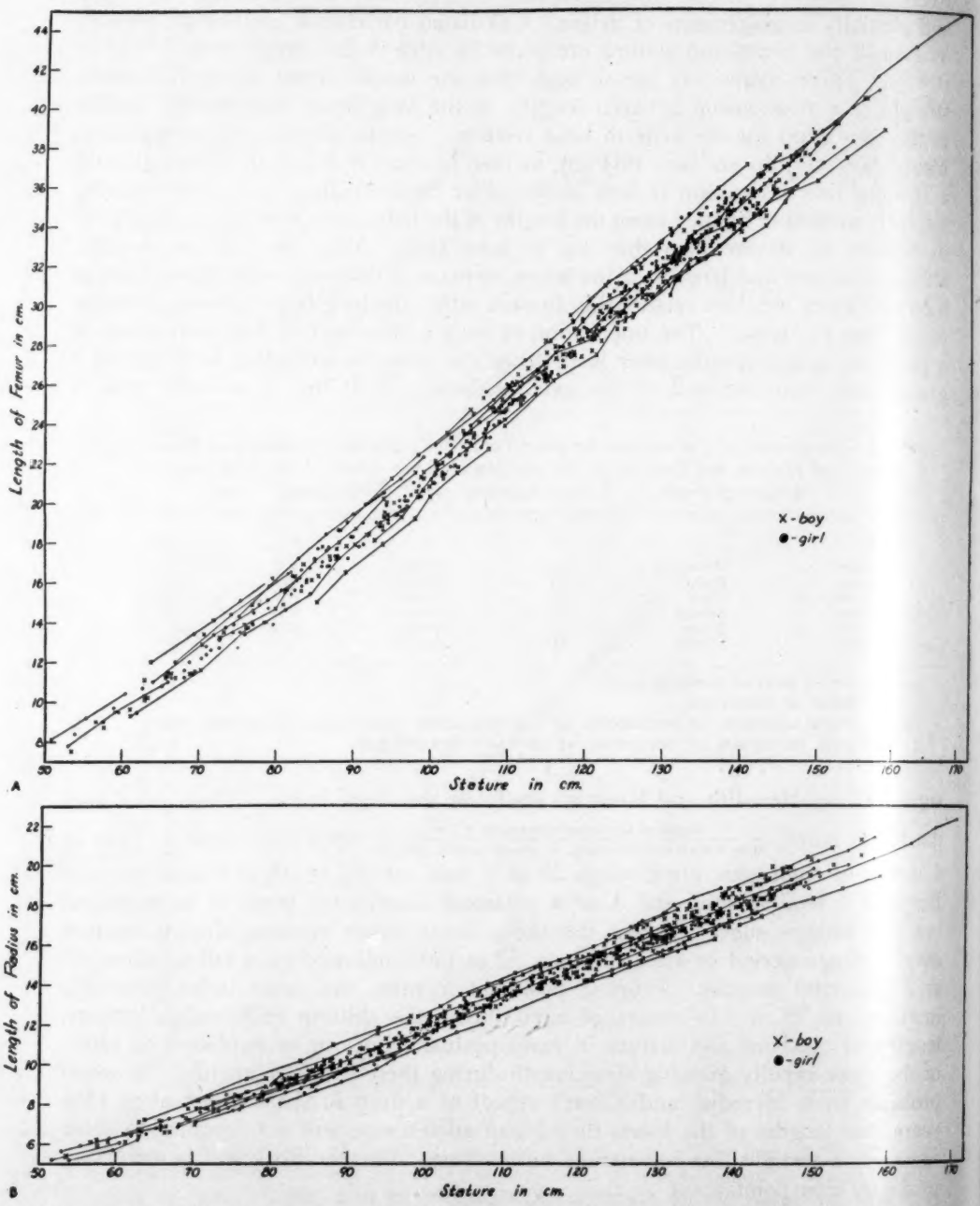



Chart 8.—Pairs of measurements of length of the femur and stature (measured as total body length or total body height) and length of radius and stature plotted against each other. The lengths of the bones were measured from 611 roentgenograms of 52 girls and 61 boys up to and including 12 years of age. The successive measurements on the same child are connected by straight lines in illustrative cases. *A*, length of the femur plotted against stature; *B*, length of radius plotted against stature.

per cent for the 20 infants measured. By 5 years the proportion of stature made up by the measured length of the femur ranges from 21.60 to 23.83 per cent, with a mean value of 22.8 per cent for the 22 children in this age group. At 10 years the range for 29 children is 23.74 per cent to 26.37 per cent, with a mean value of 25.2 per cent for the $\frac{\text{length of femur}}{\text{stature}}$ ratio. Although the measured length of the femur increases progressively faster than the stature and makes up approximately one quarter of the total height at 10 years, actually, of course, the upper part of the leg makes up more than these amounts of the stature, since the measured length of the femur is from epiphysial line to epiphysial line and the uncalcified portion of the ends and the partially calcified epiphyses of the femur are not included in the measurements. The range of variation at any one age seldom exceeds 2.5 per cent, and calculated mean values form a fairly smooth growth curve, as illustrated in chart 9, in which $\frac{\text{length of femur}}{\text{stature}}$ is referred to age. In like

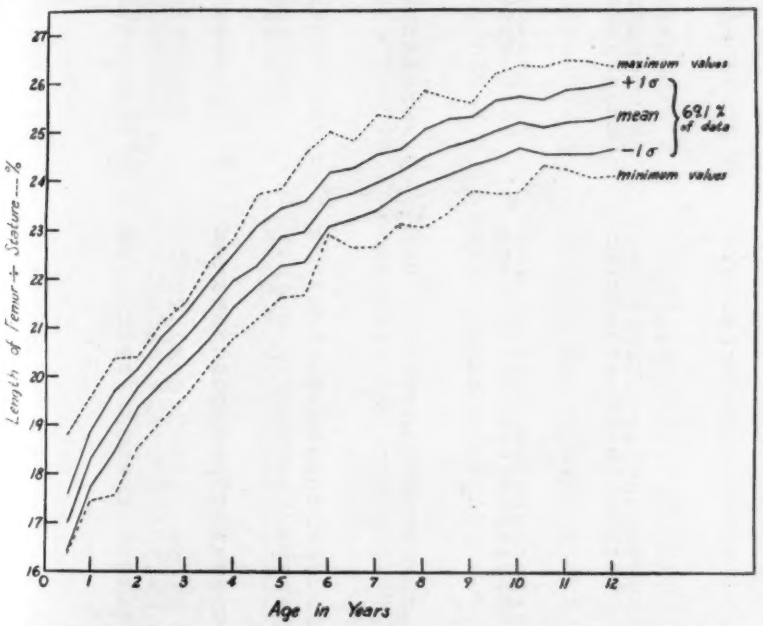



Chart 9.—Mean values, zone included in ± 1 standard deviation ($\sigma$) from the mean, and the actual range of values for $\frac{\text{length of femur}}{\text{stature}}$ ratios expressed as percentages for the 611 pairs of measurements on 52 girls and 61 boys up to and including 12 years of age.

manner, when the lengths of the femur and the tibia are added to give an estimate of the length of the leg, these values divided by stature show mean ratios of 30.6 per cent for the 6 month age group, 41.5 per cent for the 5 year group and 45.9 per cent for the 10 year group. Ratios for each six month age period have been calculated for the length of each bone to stature, as well as for length of the arm (here defined as the sum of the lengths of the humerus and the ulna) and length of the leg (the sum of the lengths of the femur and tibia) to stature. The mean values, the standard deviations, the number and percentage of values falling within ± 1 standard deviation ($\sigma$) and ± 2 standard deviations and the total range are given in table 4. No attempt has been made to smooth irregularities, and it is likely that the small number of children over 10 years of age are not as representative of the group as are the 20 or more children at each of the earlier age levels.

TABLE 4.—*Mean Values for the Ratio of Length of Each Long Bone (as Measured from the Roentgenograms of 51 Girls and 60 Boys) to Total Body Stature (Measured as Body Length or Body Height) from 6 Months to 12 Years of Age, Expressed in Percentages*

A. $\frac{\text{Length of Humerus}}{\text{Stature}} \times 100$

| Age, Years | n | Mean | σ Mean | Values Within ±1 σ from the Mean | | Values Within ±2 σ from the Mean | | Actual Range |
|---|---|---|---|---|---|---|---|---|
| | | | | No. | % | No. | % | |
| ½ | 20 | 13.45 | 0.38 | 17 | 85 | 18 | 90 | 12.19-14.25 |
| 1 | 24 | 14.22 | 0.37 | 16 | 67 | 23 | 96 | 13.62-15.00 |
| 1½ | 29 | 14.60 | 0.46 | 20 | 69 | 27 | 93 | 13.58-15.60 |
| 2 | 25 | 14.98 | 0.43 | 17 | 68 | 24 | 96 | 14.14-15.87 |
| 2½ | 24 | 15.15 | 0.45 | 17 | 71 | 23 | 96 | 13.97-15.94 |
| 3 | 25 | 15.41 | 0.48 | 16 | 64 | 25 | 100 | 14.58-16.29 |
| 3½ | 22 | 15.65 | 0.39 | 14 | 64 | 22 | 100 | 14.92-16.29 |
| 4 | 22 | 16.04 | 0.43 | 15 | 68 | 21 | 96 | 15.09-16.74 |
| 4½ | 27 | 16.23 | 0.51 | 19 | 70 | 26 | 96 | 15.22-17.34 |
| 5 | 22 | 16.51 | 0.49 | 16 | 73 | 21 | 96 | 15.36-17.45 |
| 5½ | 21 | 16.49 | 0.53 | 14 | 67 | 20 | 95 | 15.50-17.60 |
| 6 | 22 | 16.70 | 0.47 | 19 | 86 | 21 | 96 | 15.09-17.24 |
| 6½ | 25 | 16.69 | 0.48 | 18 | 72 | 23 | 92 | 15.58-17.66 |
| 7 | 32 | 16.77 | 0.53 | 22 | 69 | 31 | 97 | 15.82-18.23 |
| 7½ | 34 | 16.83 | 0.44 | 24 | 71 | 31 | 91 | 15.81-17.91 |
| 8 | 33 | 17.07 | 0.50 | 25 | 76 | 31 | 94 | 16.16-18.29 |
| 8½ | 28 | 17.10 | 0.48 | 19 | 68 | 28 | 100 | 16.23-18.05 |
| 9 | 30 | 17.20 | 0.42 | 21 | 70 | 29 | 97 | 16.19-18.02 |
| 9½ | 33 | 17.28 | 0.49 | 25 | 76 | 31 | 94 | 15.81-18.21 |
| 10 | 29 | 17.31 | 0.43 | 20 | 69 | 28 | 97 | 16.06-17.89 |
| 10½ | 21 | 17.38 | 0.42 | 15 | 71 | 20 | 95 | 16.26-17.97 |
| 11 | 15 | 17.43 | 0.41 | 12 | 80 | 14 | 93 | 16.64-18.27 |
| 11½ | 14 | 17.47 | 0.47 | 12 | 86 | 13 | 93 | 16.33-17.94 |
| 12 | 10 | 17.32 | 0.48 | 7 | 70 | 9 | 90 | 16.28-17.86 |
| Total | 587 | | | 420 | 71.5 | 559 | 95.2 | |

B. $\frac{\text{Length of Radius}}{\text{Stature}} \times 100$

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| ½ | 20 | 10.44 | 0.50 | 13 | 65 | 19 | 95 | 9.85-11.54 |
| 1 | 24 | 10.86 | 0.47 | 17 | 71 | 23 | 96 | 10.20-12.06 |
| 1½ | 29 | 10.99 | 0.46 | 21 | 72 | 27 | 93 | 9.90-12.14 |
| 2 | 25 | 11.14 | 0.45 | 17 | 68 | 24 | 96 | 10.25-12.47 |
| 2½ | 24 | 11.25 | 0.45 | 19 | 79 | 22 | 92 | 10.12-12.54 |
| 3 | 25 | 11.50 | 0.46 | 18 | 72 | 24 | 96 | 10.73-12.63 |
| 3½ | 22 | 11.63 | 0.35 | 15 | 68 | 21 | 96 | 10.93-12.36 |
| 4 | 22 | 12.01 | 0.40 | 14 | 64 | 21 | 96 | 11.14-12.68 |
| 4½ | 27 | 12.13 | 0.38 | 20 | 74 | 25 | 93 | 11.18-12.72 |
| 5 | 22 | 12.32 | 0.43 | 13 | 59 | 22 | 100 | 11.46-13.07 |
| 5½ | 21 | 12.23 | 0.41 | 16 | 76 | 20 | 95 | 11.33-12.96 |
| 6 | 22 | 12.48 | 0.34 | 13 | 59 | 22 | 100 | 11.79-13.06 |
| 6½ | 25 | 12.36 | 0.42 | 18 | 72 | 24 | 96 | 11.58-13.21 |
| 7 | 32 | 12.45 | 0.42 | 20 | 62 | 31 | 97 | 11.70-13.46 |
| 7½ | 34 | 12.48 | 0.41 | 23 | 68 | 32 | 94 | 11.69-13.44 |
| 8 | 33 | 12.61 | 0.40 | 23 | 70 | 31 | 94 | 11.81-13.50 |
| 8½ | 28 | 12.60 | 0.37 | 21 | 75 | 26 | 93 | 11.85-13.36 |
| 9 | 30 | 12.67 | 0.34 | 23 | 77 | 27 | 90 | 11.92-13.39 |
| 9½ | 33 | 12.75 | 0.40 | 22 | 67 | 32 | 97 | 11.87-13.52 |
| 10 | 29 | 12.88 | 0.39 | 18 | 62 | 28 | 97 | 12.18-13.76 |
| 10½ | 21 | 13.06 | 0.34 | 16 | 76 | 20 | 95 | 12.26-13.58 |
| 11 | 15 | 13.11 | 0.38 | 11 | 73 | 14 | 93 | 12.21-13.67 |
| 11½ | 14 | 13.18 | 0.37 | 10 | 71 | 13 | 93 | 12.23-13.72 |
| 12 | 11 | 13.15 | 0.40 | 8 | 73 | 10 | 91 | 12.25-13.63 |
| Total | 588 | | | 409 | 69.5 | 558 | 94.9 | |

C. $\frac{\text{Length of Ulna}}{\text{Stature}} \times 100$

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| ½ | 20 | 11.62 | 0.50 | 16 | 80 | 20 | 100 | 10.67-12.63 |
| 1 | 24 | 12.18 | 0.48 | 16 | 67 | 23 | 96 | 11.42-13.29 |
| 1½ | 29 | 12.38 | 0.48 | 21 | 72 | 27 | 93 | 11.32-13.57 |
| 2 | 25 | 12.50 | 0.48 | 18 | 72 | 24 | 96 | 11.65-13.94 |
| 2½ | 24 | 12.59 | 0.44 | 21 | 88 | 22 | 92 | 11.40-13.82 |
| 3 | 25 | 12.83 | 0.41 | 16 | 64 | 24 | 96 | 12.22-13.85 |
| 3½ | 22 | 12.91 | 0.33 | 16 | 73 | 21 | 96 | 12.35-13.69 |
| 4 | 22 | 13.31 | 0.37 | 15 | 68 | 22 | 100 | 12.58-13.97 |
| 4½ | 27 | 13.38 | 0.34 | 18 | 67 | 26 | 96 | 12.58-14.06 |
| 5 | 22 | 13.59 | 0.47 | 14 | 64 | 21 | 96 | 12.62-14.47 |
| 5½ | 21 | 13.48 | 0.43 | 14 | 67 | 19 | 90 | 12.61-14.44 |
| 6 | 22 | 13.64 | 0.38 | 15 | 68 | 20 | 91 | 12.79-14.45 |
| 6½ | 25 | 13.57 | 0.44 | 18 | 72 | 25 | 100 | 12.79-14.37 |
| 7 | 32 | 13.64 | 0.45 | 22 | 69 | 31 | 97 | 12.83-14.94 |
| 7½ | 34 | 13.66 | 0.45 | 25 | 74 | 33 | 97 | 12.90-14.97 |
| 8 | 33 | 13.76 | 0.43 | 25 | 76 | 32 | 97 | 12.96-14.99 |
| 8½ | 28 | 13.76 | 0.41 | 17 | 61 | 28 | 100 | 13.02-14.49 |
| 9 | 30 | 13.83 | 0.37 | 20 | 67 | 30 | 100 | 13.12-14.48 |
| 9½ | 33 | 13.92 | 0.41 | 22 | 67 | 31 | 94 | 13.03-14.57 |
| 10 | 29 | 13.99 | 0.43 | 18 | 62 | 29 | 100 | 13.13-14.80 |
| 10½ | 21 | 14.22 | 0.35 | 16 | 76 | 20 | 95 | 13.25-14.81 |
| 11 | 15 | 14.24 | 0.44 | 10 | 67 | 14 | 93 | 13.14-14.76 |
| 11½ | 14 | 14.35 | 0.39 | 11 | 78 | 13 | 93 | 13.27-14.91 |
| 12 | 11 | 14.38 | 0.39 | 9 | 82 | 10 | 91 | 13.41-14.78 |
| Total | 588 | | | 413 | 70.2 | 565 | 96.1 | |

TABLE 4.—*Mean Values for the Ratio of Length of Each Long Bone (as Measured from the Roentgenograms of 51 Girls and 60 Boys) to Total Body Stature (Measured as Body Length or Body Height) from 6 Months to 12 Years of Age, Expressed in Percentages—Continued*

| Age, Years | n | Mean | σ Mean | Values Within ± 1 σ from the Mean | | Values Within ± 2 σ from the Mean | | Actual Range |
|---|---|---|---|---|---|---|---|---|
| | | | | No. | % | No. | % | |
| | | | D. Length of Femur / Stature × 100 | | | | | |
| ½ | 20 | 17.00 | 0.58 | 16 | 80 | 19 | 95 | 16.35-18.81 |
| 1 | 24 | 18.31 | 0.56 | 16 | 67 | 23 | 96 | 17.44-19.58 |
| 1½ | 29 | 19.06 | 0.63 | 19 | 66 | 27 | 93 | 17.56-20.38 |
| 2 | 25 | 19.75 | 0.41 | 19 | 76 | 24 | 96 | 18.52-20.41 |
| 2½ | 25 | 20.33 | 0.47 | 18 | 72 | 24 | 96 | 19.08-21.10 |
| 3 | 25 | 20.78 | 0.52 | 18 | 72 | 23 | 92 | 19.57-21.51 |
| 3½ | 22 | 21.34 | 0.58 | 15 | 68 | 22 | 100 | 20.21-22.34 |
| 4 | 22 | 21.95 | 0.55 | 14 | 64 | 20 | 91 | 20.76-22.79 |
| 4½ | 27 | 22.46 | 0.62 | 16 | 59 | 24 | 89 | 21.14-23.71 |
| 5 | 22 | 22.85 | 0.58 | 13 | 59 | 21 | 96 | 21.60-23.83 |
| 5½ | 21 | 22.96 | 0.62 | 16 | 76 | 19 | 90 | 21.65-24.56 |
| 6 | 22 | 23.60 | 0.56 | 14 | 64 | 20 | 91 | 22.90-25.02 |
| 6½ | 25 | 23.73 | 0.53 | 18 | 72 | 23 | 92 | 22.64-24.82 |
| 7 | 32 | 23.96 | 0.59 | 23 | 72 | 29 | 91 | 22.65-25.36 |
| 7½ | 34 | 24.19 | 0.46 | 25 | 74 | 32 | 94 | 23.11-25.27 |
| 8 | 33 | 24.48 | 0.57 | 24 | 73 | 31 | 94 | 23.04-25.86 |
| 8½ | 28 | 24.68 | 0.58 | 20 | 71 | 27 | 96 | 23.32-25.70 |
| 9 | 30 | 24.81 | 0.50 | 20 | 67 | 29 | 97 | 23.77-25.60 |
| 9½ | 33 | 25.04 | 0.61 | 23 | 70 | 31 | 94 | 23.72-26.20 |
| 10 | 29 | 25.20 | 0.53 | 21 | 72 | 27 | 93 | 23.74-26.37 |
| 10½ | 21 | 25.10 | 0.56 | 13 | 62 | 20 | 95 | 24.30-26.34 |
| 11 | 15 | 25.19 | 0.67 | 9 | 60 | 15 | 100 | 24.22-26.47 |
| 11½ | 14 | 25.21 | 0.68 | 9 | 64 | 14 | 100 | 24.05-26.45 |
| 12 | 11 | 25.32 | 0.68 | 7 | 64 | 11 | 100 | 24.07-26.35 |
| Total | 589 | | | 406 | 69.1 | 555 | 94.2 | |
| | | | E. Length of Tibia / Stature × 100 | | | | | |
| ½ | 20 | 13.59 | 0.42 | 14 | 70 | 19 | 95 | 12.86-14.48 |
| 1 | 24 | 14.79 | 0.45 | 15 | 62 | 23 | 96 | 13.82-15.61 |
| 1½ | 29 | 15.42 | 0.45 | 22 | 76 | 28 | 96 | 14.27-16.24 |
| 2 | 25 | 16.11 | 0.43 | 17 | 68 | 24 | 96 | 15.26-17.12 |
| 2½ | 25 | 16.55 | 0.43 | 17 | 68 | 23 | 92 | 15.67-17.53 |
| 3 | 26 | 17.02 | 0.55 | 19 | 73 | 25 | 96 | 15.42-17.92 |
| 3½ | 22 | 17.37 | 0.56 | 15 | 68 | 21 | 96 | 15.87-18.24 |
| 4 | 22 | 17.90 | 0.65 | 18 | 82 | 20 | 91 | 16.22-19.21 |
| 4½ | 27 | 18.24 | 0.58 | 20 | 74 | 26 | 96 | 16.68-19.14 |
| 5 | 22 | 18.66 | 0.66 | 17 | 77 | 21 | 96 | 16.88-19.82 |
| 5½ | 21 | 18.62 | 0.68 | 17 | 81 | 18 | 86 | 17.01-20.02 |
| 6 | 22 | 19.14 | 0.48 | 15 | 68 | 21 | 96 | 18.29-20.13 |
| 6½ | 25 | 19.22 | 0.65 | 18 | 72 | 23 | 92 | 17.72-20.75 |
| 7 | 32 | 19.39 | 0.68 | 22 | 69 | 29 | 91 | 17.79-20.89 |
| 7½ | 34 | 19.58 | 0.62 | 25 | 74 | 31 | 91 | 18.14-21.22 |
| 8 | 33 | 19.88 | 0.65 | 22 | 67 | 31 | 94 | 18.22-21.36 |
| 8½ | 28 | 19.99 | 0.56 | 19 | 68 | 27 | 96 | 18.45-21.01 |
| 9 | 30 | 20.18 | 0.61 | 21 | 70 | 29 | 97 | 18.59-21.16 |
| 9½ | 33 | 20.37 | 0.67 | 23 | 70 | 31 | 94 | 18.57-21.45 |
| 10 | 29 | 20.66 | 0.64 | 17 | 59 | 28 | 97 | 19.34-21.60 |
| 10½ | 21 | 20.87 | 0.70 | 15 | 71 | 20 | 95 | 19.45-22.08 |
| 11 | 15 | 21.03 | 0.69 | 9 | 60 | 14 | 93 | 19.55-21.91 |
| 11½ | 14 | 20.96 | 0.80 | 9 | 64 | 13 | 93 | 19.52-22.08 |
| 12 | 10 | 21.10 | 0.74 | 4 | 40 | 10 | 100 | 19.84-21.96 |
| Total | 589 | | | 410 | 69.6 | 555 | 94.2 | |
| | | | F. Length of Fibula / Stature × 100 | | | | | |
| ½ | 20 | 12.93 | 0.40 | 15 | 75 | 18 | 90 | 12.43-14.03 |
| 1 | 24 | 14.25 | 0.49 | 16 | 67 | 24 | 100 | 13.27-15.21 |
| 1½ | 29 | 15.04 | 0.52 | 21 | 72 | 27 | 93 | 13.53-16.12 |
| 2 | 25 | 15.77 | 0.54 | 18 | 72 | 23 | 92 | 14.67-17.12 |
| 2½ | 25 | 16.33 | 0.46 | 20 | 80 | 24 | 96 | 15.24-17.43 |
| 3 | 26 | 16.90 | 0.57 | 16 | 61 | 24 | 92 | 15.53-17.82 |
| 3½ | 22 | 17.23 | 0.55 | 16 | 73 | 21 | 96 | 15.98-18.04 |
| 4 | 22 | 17.88 | 0.76 | 16 | 73 | 21 | 96 | 16.32-19.21 |
| 4½ | 27 | 18.14 | 0.63 | 20 | 74 | 25 | 93 | 16.63-19.04 |
| 5 | 22 | 18.59 | 0.67 | 17 | 77 | 21 | 96 | 16.78-19.81 |
| 5½ | 21 | 18.54 | 0.66 | 15 | 71 | 19 | 90 | 17.01-19.85 |
| 6 | 22 | 19.04 | 0.46 | 14 | 64 | 21 | 96 | 18.38-20.13 |
| 6½ | 25 | 19.08 | 0.64 | 17 | 68 | 23 | 92 | 17.46-20.52 |
| 7 | 32 | 19.27 | 0.66 | 23 | 72 | 29 | 91 | 17.63-20.63 |
| 7½ | 34 | 19.42 | 0.60 | 26 | 76 | 31 | 91 | 17.90-21.07 |
| 8 | 33 | 19.69 | 0.66 | 22 | 67 | 30 | 91 | 18.07-21.14 |
| 8½ | 28 | 19.82 | 0.57 | 19 | 68 | 27 | 96 | 18.30-20.82 |
| 9 | 30 | 19.98 | 0.61 | 20 | 67 | 29 | 97 | 18.44-21.12 |
| 9½ | 33 | 20.16 | 0.62 | 21 | 64 | 32 | 97 | 18.35-21.24 |
| 10 | 29 | 20.42 | 0.64 | 17 | 59 | 29 | 100 | 19.27-21.43 |
| 10½ | 21 | 20.55 | 0.66 | 13 | 62 | 21 | 100 | 19.37-21.52 |
| 11 | 15 | 20.68 | 0.70 | 9 | 60 | 15 | 100 | 19.45-21.83 |
| 11½ | 14 | 20.63 | 0.75 | 8 | 57 | 14 | 100 | 19.60-21.86 |
| 12 | 10 | 20.76 | 0.70 | 5 | 50 | 10 | 100 | 19.65-21.86 |
| Total | 589 | | | 404 | 68.6 | 558 | 94.7 | |

TABLE 4.—*Mean Values for the Ratio of Length of Each Long Bone (As Measured from the Roentgenograms of 51 Girls and 60 Boys) to Total Body Stature (Measured as Body Length or Body Height) from 6 Months to 12 Years of Age, Expressed as Percentages—Continued*

| Age, Years | n | Mean | σ Mean | Values Within ±1 σ from the Mean | | Values Within ±2 σ from the Mean | | Actual Range |
|---|---|---|---|---|---|---|---|---|
| | | | | No. | % | No. | % | |
| | | | G. $\frac{\text{Length of Humerus + Ulna}}{\text{Stature}} \times 100$ | | | | | |
| ½ | 20 | 25.07 | 0.56 | 12 | 60 | 19 | 95 | 24.25-26.27 |
| 1 | 24 | 26.40 | 0.76 | 15 | 62 | 23 | 96 | 25.13-28.08 |
| 1½ | 29 | 26.98 | 0.89 | 20 | 69 | 27 | 93 | 25.06-28.69 |
| 2 | 25 | 27.48 | 0.81 | 19 | 76 | 24 | 96 | 25.92-29.82 |
| 2½ | 24 | 27.74 | 0.78 | 18 | 75 | 23 | 96 | 26.33-29.76 |
| 3 | 25 | 28.24 | 0.84 | 17 | 68 | 24 | 96 | 26.81-29.94 |
| 3½ | 22 | 28.57 | 0.68 | 13 | 59 | 22 | 100 | 27.30-29.93 |
| 4 | 22 | 29.35 | 0.74 | 15 | 68 | 22 | 100 | 27.94-30.54 |
| 4½ | 27 | 29.61 | 0.77 | 19 | 70 | 26 | 96 | 27.96-31.13 |
| 5 | 22 | 30.10 | 0.89 | 15 | 68 | 21 | 95 | 28.28-31.58 |
| 5½ | 21 | 29.97 | 0.88 | 15 | 71 | 20 | 95 | 28.15-31.71 |
| 6 | 22 | 30.35 | 0.74 | 16 | 73 | 21 | 95 | 28.36-31.51 |
| 6½ | 25 | 30.26 | 0.84 | 17 | 68 | 25 | 100 | 28.69-31.90 |
| 7 | 32 | 30.41 | 0.91 | 22 | 69 | 31 | 97 | 28.66-33.18 |
| 7½ | 34 | 30.50 | 0.81 | 25 | 74 | 31 | 91 | 28.79-32.62 |
| 8 | 33 | 30.83 | 0.86 | 24 | 73 | 31 | 94 | 29.12-33.18 |
| 8½ | 28 | 30.86 | 0.82 | 21 | 75 | 27 | 96 | 29.40-32.54 |
| 9 | 30 | 31.03 | 0.66 | 22 | 73 | 29 | 97 | 29.31-32.27 |
| 9½ | 33 | 31.20 | 0.84 | 24 | 73 | 31 | 94 | 28.85-32.69 |
| 10 | 29 | 31.30 | 0.76 | 19 | 66 | 28 | 96 | 29.19-32.50 |
| 10½ | 21 | 31.62 | 0.71 | 16 | 76 | 20 | 95 | 29.51-32.78 |
| 11 | 15 | 31.67 | 0.75 | 12 | 80 | 14 | 93 | 29.89-32.77 |
| 11½ | 14 | 31.82 | 0.76 | 11 | 78 | 13 | 93 | 29.80-32.80 |
| 12 | 10 | 31.74 | 0.78 | 8 | 80 | 9 | 90 | 30.04-32.47 |
| Total | 587 | | | 415 | 70.7 | 561 | 95.6 | |
| | | | H. $\frac{\text{Length of Femur + Tibia}}{\text{Stature}} \times 100$ | | | | | |
| ½ | 20 | 30.59 | 0.80 | 16 | 80 | 18 | 90 | 29.32-32.92 |
| 1 | 24 | 33.09 | 0.99 | 16 | 67 | 23 | 96 | 31.45-35.14 |
| 1½ | 29 | 34.48 | 0.99 | 22 | 76 | 27 | 93 | 32.20-36.38 |
| 2 | 25 | 35.86 | 0.74 | 20 | 80 | 23 | 92 | 33.78-37.53 |
| 2½ | 25 | 36.88 | 0.81 | 18 | 72 | 24 | 96 | 34.75-38.20 |
| 3 | 25 | 37.80 | 0.95 | 20 | 80 | 23 | 92 | 35.02-39.30 |
| 3½ | 22 | 38.71 | 1.01 | 15 | 68 | 21 | 96 | 36.08-40.25 |
| 4 | 22 | 39.85 | 1.11 | 16 | 73 | 21 | 96 | 36.98-42.00 |
| 4½ | 27 | 40.70 | 1.08 | 22 | 81 | 26 | 96 | 37.86-42.46 |
| 5 | 22 | 41.51 | 1.11 | 15 | 68 | 21 | 96 | 38.48-43.58 |
| 5½ | 21 | 41.58 | 1.19 | 16 | 76 | 19 | 90 | 38.66-43.89 |
| 6 | 22 | 42.74 | 0.89 | 14 | 64 | 22 | 100 | 41.28-44.52 |
| 6½ | 25 | 42.95 | 1.08 | 16 | 64 | 24 | 96 | 40.36-44.89 |
| 7 | 32 | 43.34 | 1.11 | 22 | 69 | 30 | 94 | 40.44-45.65 |
| 7½ | 34 | 43.77 | 0.92 | 23 | 68 | 32 | 94 | 41.61-46.07 |
| 8 | 33 | 44.36 | 1.10 | 23 | 70 | 30 | 91 | 41.60-46.68 |
| 8½ | 28 | 44.64 | 0.95 | 20 | 71 | 27 | 96 | 42.19-46.42 |
| 9 | 30 | 44.99 | 0.91 | 21 | 70 | 28 | 93 | 42.80-46.57 |
| 9½ | 33 | 45.42 | 1.12 | 21 | 64 | 31 | 94 | 42.32-47.06 |
| 10 | 29 | 45.86 | 0.97 | 20 | 69 | 28 | 97 | 43.09-47.34 |
| 10½ | 21 | 45.97 | 1.16 | 14 | 67 | 21 | 100 | 43.92-48.15 |
| 11 | 15 | 46.22 | 1.25 | 9 | 60 | 15 | 100 | 43.76-47.92 |
| 11½ | 14 | 46.17 | 1.37 | 9 | 64 | 14 | 100 | 44.10-47.90 |
| 12 | 10 | 46.39 | 1.46 | 7 | 70 | 10 | 100 | 44.33-47.83 |
| Total | 588 | | | 415 | 70.6 | 558 | 94.9 | |

Since we now have set up provisional standards for lengths of the bones at different ages and have also established the approximate range of variation for the relationship of these lengths to stature, it becomes relatively simple to ascertain for a given child how his bone lengths for stature and bone lengths for age compare with those of the group. For example, the girl whose individual graphs are illustrated in chart 3 *D* is one of the 2 shortest girls in the series at each age level from 5 years 9 months through 10 years. However, the lengths of her leg bones are sufficiently high so that the $\frac{\text{femur}}{\text{stature}}$ ratio is well above average for her age and the $\frac{\text{femur + tibia}}{\text{stature}}$ ratio is the greatest in the group for her stature and near the upper limit of the group for her age. Thus, her low level for stature can be partially explained by her unusually short stem length.

Although the correlation coefficients for the length of one bone to that of another are extremely high (table 2) and scatter charts indicate a close relationship between the lengths of any two of the long bones throughout childhood (chart 7), the ratios between these lengths are actually changing with advancing age through childhood. For example, the mean values for the $\frac{\text{femur}}{\text{humerus}}$ ratio, which is, in a sense, an expression of the length of the upper part of the leg as compared with the length of the upper part of the arm, are 1.29 : 1 at 1 year, 1.42 : 1 at 6 years and 1.46 : 1 at 12 years of age, as might be expected from the greater growth rate of the lower extremity. While the femur is only about one and one-fourth times as long as the humerus at 1 year of age, it is nearly one and one-half times as long at 12 years. At each age interval the range is fairly large but the values are grouped so closely about the mean that approximately 75 per cent of the values fall within $\pm$ 1 standard deviation from the mean. It is probable that the figures obtained by dividing one length by another are less reliable and less significant than, for example, the relation of the length of a bone to stature. Marked deviations from the usual body proportions are more readily apparent when one compares bone lengths with the percentile standards than when one uses mean ratios.[7]

## SUMMARY AND CONCLUSIONS

Seriatim roentgenograms of the left arm and leg have been taken on a group of 113 children, 52 girls and 61 boys, at semiyearly intervals up to 12 years of age. Technical factors have been kept as constant as is possible with children of different ages. Six hundred and seventy-one sets of such roentgenograms are now available for study. From these roentgenograms, the lengths of the humerus, radius, ulna, femur, tibia and fibula have been measured. These values have been used, without correction for distortion, in the study of rates of growth of the bones and of correlations between the lengths of the bones and skeletal growth.

Increase in length of the bones on successive measurements indicate that the growth rate, at least up to 10 years of age, is remarkably orderly. Without an attempt being made to set up separate standards for boys and girls, it has been possible to set up zones of variation within the group so that the growth in length of the bones of one child could be compared with the standards. Values are given in table 1 for the tenth, twenty-fifth, fiftieth, seventy-fifth and ninetieth percentiles for each of the long bones at six month intervals from 6 months to 12 years of age.

Study of the growth of the bones of each child by comparing his growth curves with the percentile standards demonstrates that in half the children (51 per cent) there has been no significant shift in the relative position (percentile ranking) of any of the six bones during the period over which measurements have been made. Sample graphs of this group are given in chart 3. A second group (15 per cent) shows either increase or lowering of their relative position on successive measurements of at least five of the six bones. Chart 4 is illustrative of this group. A third group (27 per cent) with lowering or improvement of the relative position of one, two, three or four bones and no change in the others is illustrated in chart 5. A fourth, smaller, group (7 per cent), illustrated in chart 6, shows less consistency in the rates of growth of the bones, with some elevation of relative position of one or more bones and some lowering of relative position of one or more bones, along with at least one bone which has grown at the group rate and therefore shows no change in percentile ranking on successive measurements.

7. For this reason the mean ratios have not been included in this paper although they are available.

TABLE 5.—*Individual Data*

A. Boys

| Subject No. | Age | | Stature, Cm. | Lengths of Bones, Cm. | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Yr. | Mo. | | Humerus | Radius | Ulna | Femur | Tibia | Fibula |
| 1 | 0 | 1 | 53.5 | 6.6 | 5.4 | 6.2 | 7.55 | 6.3 | 5.9 |
| | 3 | 3 | 97.0 | 14.2 | 11.1 | 12.1 | 19.8 | 16.3 | 16.3 |
| | 4 | 0 | 101.4 | 15.3 | 12.0 | 13.2 | 22.0 | 17.9 | 17.95 |
| | 4 | 6 | 104.5 | 15.9 | 12.5 | 13.8 | 22.7 | 18.7 | 18.8 |
| | 5 | 0 | ..... | 16.5 | 13.05 | 14.4 | 24.1 | 19.6 | 19.8 |
| | 5 | 6 | 111.6 | 17.3 | 13.6 | 15.0 | 25.4 | 20.5 | 20.6 |
| | 6 | 0 | 115.3 | 17.4 | 14.1 | 15.3 | 26.4 | 21.2 | 21.3 |
| | 6 | 6 | 119.4 | 18.6 | 14.6 | 16.0 | 27.4 | 22.1 | 22.3 |
| 2 | 0 | 2 | 56.7 | 7.7 | 6.1 | 7.0 | 9.0 | 7.3 | 6.8 |
| 3 | 0 | 2 | 57.6 | 7.4 | 6.2 | 6.9 | 9.0 | 7.4 | 7.1 |
| | 0 | 4 | 63.0 | 8.35 | 6.7 | 7.5 | 10.1 | 8.1 | 7.7 |
| | 0 | 6 | 66.0 | 9.0 | 7.15 | 8.0 | 11.3 | 8.7 | 8.5 |
| 4 | 0 | 3 | 61.1 | 8.0 | 6.5 | 7.1 | 9.65 | 8.1 | 7.6 |
| | 1 | 0 | 76.0 | 10.75 | 8.4 | 9.2 | 13.9 | 11.2 | 10.9 |
| | 1 | 3 | 79.5 | 11.7 | 8.9 | 9.8 | 14.7 | 12.05 | 11.85 |
| | 1 | 6 | 83.3 | 12.3 | 9.3 | 10.35 | 15.55 | 13.1 | 12.8 |
| | 2 | 0 | 87.8 | 13.1 | 9.9 | 10.95 | 17.2 | 14.5 | 14.25 |
| | 2 | 6 | 91.7 | 13.9 | 10.45 | 11.5 | 18.4 | 15.5 | 15.1 |
| | 3 | 0 | 94.4 | 14.8 | 11.0 | 12.1 | 19.4 | 16.7 | 16.5 |
| | 3 | 6 | 99.1 | 15.3 | 11.5 | 12.5 | 20.6 | 17.55 | 17.4 |
| 5 | 0 | 3 | 61.2 | 7.9 | 6.1 | 7.0 | 9.3 | 7.8 | 7.0 |
| | 0 | 6 | 70.3 | 9.45 | 7.0 | 7.85 | 11.6 | 9.7 | 8.8 |
| | 1 | 0 | 76.4 | 10.8 | 7.9 | 8.8 | 13.75 | 11.4 | 10.5 |
| | 1 | 6 | 81.2 | 12.1 | 8.8 | 9.8 | 15.5 | 12.8 | 12.0 |
| | 2 | 0 | 85.1 | 13.25 | 9.4 | 10.4 | 17.1 | 14.1 | 13.4 |
| | 2 | 6 | 90.3 | 14.2 | 10.1 | 11.1 | 18.7 | 15.2 | 14.6 |
| | 3 | 0 | 94.3 | 14.9 | 10.6 | 11.7 | 20.0 | 16.2 | 15.65 |
| 6 | 0 | 3 | 57.7 | 7.0 | 5.9 | 6.75 | 8.7 | 7.5 | 7.0 |
| | 0 | 6 | 63.0 | 8.4 | 6.4 | 7.3 | 10.3 | 8.5 | 8.0 |
| | 1 | 0 | 70.5 | 10.2 | 7.8 | 8.9 | 12.9 | 10.6 | 10.0 |
| | 1 | 6 | 77.0 | 11.4 | 8.6 | 9.7 | 14.9 | 11.9 | 11.5 |
| | 2 | 0 | 82.5 | 12.5 | 9.3 | 10.4 | 16.3 | 13.2 | 12.7 |
| | 2 | 6 | 86.0 | 13.35 | 9.9 | 11.0 | 17.8 | 14.25 | 13.8 |
| 7 | 0 | 4 | 63.3 | 8.0 | 6.6 | 7.25 | 10.2 | 8.2 | 7.7 |
| | 0 | 6 | 67.0 | 9.1 | 6.6 | 7.15 | 11.2 | 9.0 | 8.4 |
| 8 | 0 | 6 | 63.8 | 8.4 | 7.2 | 7.9 | 12.0 | 9.0 | 8.1 |
| | 1 | 0 | 72.0 | 10.8 | 8.4 | 9.2 | 14.1 | 11.2 | 10.4 |
| | 1 | 6 | 79.5 | 12.4 | 9.3 | 10.4 | 16.2 | 12.6 | 11.95 |
| 9 | 0 | 6 | 69.5 | 9.3 | 7.2 | 8.2 | 11.5 | 9.6 | 9.1 |
| | 1 | 0 | 78.8 | 11.05 | 8.4 | 9.5 | 14.0 | 11.3 | 11.15 |
| | 1 | 6 | 84.7 | 12.2 | 9.3 | 10.4 | 16.1 | 12.9 | 12.8 |
| | 2 | 0 | 89.2 | 13.2 | 10.0 | 11.25 | 17.8 | 14.4 | 14.3 |
| | 2 | 6 | 94.1 | 14.1 | 10.6 | 11.9 | 19.5 | 15.7 | 15.7 |
| | 3 | 0 | 97.4 | 15.0 | 11.2 | 12.6 | 20.4 | 16.7 | 16.6 |
| 10 | 0 | 6 | 68.9 | 8.4 | 7.95 | 8.7 | 11.4 | 9.8 | 9.5 |
| | 1 | 0 | 77.1 | 11.4 | 9.3 | 10.25 | 14.0 | 11.9 | 11.7 |
| | 1 | 6 | 84.0 | 12.7 | 10.2 | 11.4 | 16.0 | 13.6 | 13.4 |
| | 2 | 0 | 88.2 | 14.0 | 11.0 | 12.3 | 18.0 | 15.1 | 15.1 |
| | 2 | 6 | 94.1 | 15.0 | 11.8 | 13.0 | 19.3 | 16.5 | 16.4 |
| | 3 | 0 | 98.2 | 15.8 | 12.4 | 13.6 | 20.55 | 17.6 | 17.5 |
| 11 | 0 | 6 | 66.4 | 8.65 | 6.6 | 7.5 | 11.4 | 8.8 | 8.3 |
| | 1 | 0 | 73.3 | 10.5 | 7.7 | 8.8 | 13.5 | 10.6 | 10.1 |
| 12 | 0 | 6 | 63.0 | 8.6 | 7.1 | 7.95 | 11.1 | 8.6 | 8.3 |
| | 1 | 0 | 70.7 | 10.3 | 8.2 | 9.1 | 13.4 | 11.0 | 10.6 |
| 13 | 0 | 6 | 69.2 | 9.4 | 7.3 | 8.05 | 11.4 | 8.9 | 8.6 |
| | 1 | 0 | 78.5 | 10.8 | 8.3 | 9.2 | 14.0 | 11.2 | 11.0 |
| 14 | 0 | 9 | 72.7 | 10.3 | 8.1 | 8.8 | 13.2 | 10.4 | 9.9 |
| | 1 | 3 | 80.1 | 11.7 | 9.0 | 10.3 | 14.7 | 12.3 | 12.0 |
| | 1 | 6 | 81.5 | 12.2 | 9.5 | 10.55 | 15.6 | 12.6 | 12.4 |
| | 2 | 0 | 87.0 | 13.3 | 10.2 | 11.45 | 17.3 | 14.3 | 14.2 |
| | 2 | 6 | .... | 14.5 | 11.1 | 12.1 | 18.9 | 15.75 | 15.7 |
| | 3 | 0 | 93.9 | 15.3 | 11.6 | 12.8 | 20.2 | 16.7 | 16.6 |
| | 3 | 6 | 97.9 | 15.9 | 12.1 | 13.4 | 21.5 | 17.8 | 17.65 |
| 15 | 1 | 1 | 81.0 | 11.5 | 9.0 | 10.3 | 14.9 | 12.2 | 11.5 |
| | 1 | 6 | 85.6 | 12.5 | 9.7 | 11.0 | 16.3 | 13.5 | 12.6 |
| | 2 | 0 | 87.8 | 13.0 | 10.2 | 11.5 | 17.7 | 14.4 | 13.7 |
| 16 | 1 | 6 | 85.4 | 11.6 | 8.9 | 9.8 | 15.0 | 12.5 | 12.0 |
| | 2 | 0 | 89.1 | 12.6 | 9.6 | 10.5 | 16.5 | 13.6 | 13.1 |
| | 2 | 6 | 93.8 | 13.5 | 10.3 | 11.4 | 17.9 | 14.7 | 14.3 |
| | 3 | 0 | 98.1 | 14.3 | 10.9 | 12.0 | 19.2 | 15.7 | 15.3 |
| | 3 | 6 | 99.9 | 15.1 | 11.5 | 12.6 | 20.3 | 16.6 | 16.2 |
| | 4 | 0 | 103.4 | 15.9 | 12.1 | 13.4 | 21.5 | 17.5 | 17.15 |
| | 4 | 6 | 107.4 | 16.6 | 12.7 | 14.0 | 22.7 | 18.5 | 18.1 |

TABLE 5.—*Individual Data—Continued*

A. Boys—Continued

| Subject No. | Age | | Stature, Cm. | Lengths of Bones, Cm. | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Yr. | Mo. | | Humerus | Radius | Ulna | Femur | Tibia | Fibula |
| 17 | 2 | 0 | 90.3 | 13.8 | 10.6 | 11.6 | 17.9 | 15.0 | 15.0 |
| | 2 | 6 | 95.7 | .... | .... | .... | 19.4 | 16.1 | 16.3 |
| | 3 | 0 | 100.3 | 15.9 | 12.0 | 13.15 | 21.05 | 17.4 | 17.6 |
| | 3 | 6 | ..... | 16.9 | 12.7 | 13.8 | 22.4 | 18.5 | 18.6 |
| | 4 | 0 | 105.1 | 17.6 | 13.3 | 14.5 | 23.7 | 19.4 | 19.6 |
| | 4 | 6 | 110.5 | 18.4 | 13.8 | 15.1 | 25.2 | 20.5 | 20.7 |
| | 5 | 0 | 113.8 | 19.3 | 14.4 | 15.6 | 26.3 | 21.4 | 21.6 |
| 18 | 2 | 0 | 83.6 | 12.7 | 9.4 | 10.55 | 16.9 | 13.6 | 13.25 |
| | 2 | 6 | 88.2 | 13.6 | 10.0 | 11.3 | 18.55 | 14.9 | 14.65 |
| | 3 | 0 | 93.0 | 14.45 | 10.7 | 12.0 | .... | 16.0 | 15.9 |
| | 3 | 6 | 94.9 | 15.2 | 11.3 | 12.7 | 21.2 | 17.0 | 16.8 |
| | 4 | 0 | 98.3 | 16.05 | 11.8 | 13.2 | 22.4 | 18.0 | 17.8 |
| | 4 | 6 | 102.0 | 16.55 | 12.4 | 13.85 | 23.5 | 19.0 | 18.8 |
| | 5 | 0 | 105.0 | 17.6 | 12.85 | 14.3 | 24.7 | 19.65 | 19.5 |
| 19 | 2 | 3 | 92.1 | 13.9 | 10.8 | 12.0 | 18.4 | 15.2 | 15.05 |
| | 3 | 1 | 98.4 | 15.3 | 11.8 | 13.1 | 20.3 | 16.9 | 16.8 |
| | 3 | 6 | 101.4 | 16.05 | 12.3 | 13.7 | 21.4 | 17.9 | 17.8 |
| | 4 | 0 | 103.1 | 16.9 | 12.9 | 14.4 | 22.9 | 18.9 | 18.8 |
| | 4 | 6 | 106.9 | 17.6 | 13.6 | 15.0 | 23.7 | 20.1 | 19.8 |
| | 5 | 0 | 109.7 | 18.5 | 14.1 | 15.6 | 24.7 | 21.1 | 20.8 |
| 20 | 2 | 6 | 95.3 | 14.6 | 10.95 | 12.1 | 20.0 | 16.4 | 16.0 |
| | 3 | 0 | 99.0 | 15.15 | 11.5 | 12.7 | 21.2 | 17.5 | 17.1 |
| | 3 | 6 | 102.5 | 15.9 | 12.0 | 13.2 | 22.5 | 18.4 | 18.1 |
| | 4 | 0 | 104.3 | 16.8 | 12.5 | 13.8 | 23.5 | 19.1 | 19.0 |
| | 4 | 6 | 107.6 | 17.6 | 12.9 | 14.1 | 24.8 | 20.1 | 19.9 |
| | 5 | 0 | 110.8 | 18.5 | 13.5 | 14.7 | 26.0 | 21.0 | 20.75 |
| | 5 | 6 | 114.7 | 18.9 | 13.9 | 15.2 | 27.1 | 22.0 | 21.7 |
| | 6 | 0 | 117.8 | 19.8 | 14.5 | 15.8 | 28.0 | 22.8 | 22.4 |
| 21 | 2 | 6 | 92.8 | 13.9 | 10.7 | 11.9 | 18.1 | 15.3 | 15.3 |
| | 3 | 0 | 96.3 | 14.6 | 11.3 | 12.6 | 19.3 | 16.3 | 16.4 |
| | 4 | 0 | 100.6 | 16.3 | 12.5 | 13.9 | 21.5 | 18.4 | 18.6 |
| | 4 | 6 | 105.2 | 17.0 | 13.1 | 14.5 | 22.9 | 19.3 | 19.5 |
| | 5 | 0 | 109.5 | 18.0 | 13.7 | 15.25 | 24.2 | 20.5 | 20.6 |
| | 5 | 6 | 113.5 | 18.9 | 14.3 | 15.9 | 25.5 | 21.5 | 21.6 |
| 22 | 2 | 6 | 93.7 | 14.2 | 10.8 | 12.0 | 18.8 | 15.2 | 15.3 |
| | 3 | 0 | 97.0 | 14.8 | 11.4 | 12.7 | 20.1 | 16.0 | 16.2 |
| | 3 | 6 | 101.6 | 15.7 | 11.9 | 13.2 | 21.2 | 16.9 | 17.1 |
| | 4 | 0 | 102.4 | 16.5 | 12.4 | 13.8 | 22.4 | 17.7 | 17.9 |
| | 4 | 6 | 105.4 | 16.8 | 13.0 | 14.5 | 23.5 | 18.7 | 18.8 |
| | 5 | 0 | 109.1 | 17.8 | 13.5 | 15.0 | 24.6 | 19.4 | 19.5 |
| | 5 | 6 | 111.8 | 18.6 | 13.9 | 15.5 | 25.2 | 20.1 | 20.2 |
| | 6 | 0 | 114.8 | 18.9 | 14.4 | 15.9 | 26.4 | 21.0 | 21.1 |
| 23 | 3 | 0 | 96.4 | .... | .... | .... | 19.6 | 17.0 | 17.0 |
| | 3 | 6 | 99.8 | 15.8 | 11.9 | 12.9 | 20.8 | 18.2 | 18.0 |
| | 4 | 0 | 100.7 | 16.4 | 12.4 | 13.5 | 21.9 | 19.0 | 18.9 |
| | 4 | 6 | 104.5 | 17.2 | 12.9 | 14.1 | 23.1 | 20.0 | 19.9 |
| | 5 | 0 | 108.8 | 17.9 | 13.4 | 14.7 | 24.2 | 21.0 | 20.8 |
| | 5 | 6 | 111.2 | 18.8 | 14.0 | 15.3 | 25.2 | 21.8 | 21.65 |
| | 6 | 0 | 114.9 | 19.5 | 14.6 | 16.0 | 26.4 | 22.7 | 22.6 |
| | 6 | 6 | 118.5 | 19.9 | 15.1 | 16.5 | 27.6 | 23.8 | 23.5 |
| | 7 | 0 | 122.1 | 21.0 | 15.6 | 16.9 | 28.8 | 24.7 | 24.4 |
| | 7 | 6 | 126.4 | 21.6 | 16.15 | 17.7 | 30.0 | 25.5 | 25.2 |
| 24 | 3 | 3 | .... | 15.3 | 11.7 | 12.9 | 20.0 | 16.2 | 16.6 |
| | 4 | 2 | 99.1 | 16.2 | 12.4 | 13.7 | 21.5 | 17.5 | 17.95 |
| | 4 | 6 | 101.9 | 16.8 | 12.9 | 14.15 | 22.4 | 18.3 | 18.6 |
| | 5 | 1 | 104.4 | 17.7 | 13.5 | 14.9 | 23.5 | 19.1 | 19.4 |
| | 5 | 6 | 108.0 | 18.5 | 14.0 | 15.6 | 24.6 | 20.1 | 20.4 |
| | 6 | 0 | 111.4 | 19.0 | 14.55 | 16.1 | 25.9 | 21.0 | 21.2 |
| | 6 | 6 | 114.8 | 19.25 | 15.0 | 16.5 | 26.8 | 21.9 | 22.0 |
| 25 | 3 | 6 | 98.7 | 15.3 | 11.4 | 12.6 | 21.3 | 16.9 | 16.9 |
| | 4 | 0 | 100.5 | 15.8 | 12.0 | 13.1 | 22.1 | 17.8 | 17.8 |
| | 4 | 7 | 102.5 | 16.7 | 12.65 | 13.7 | 23.3 | 18.6 | 18.6 |
| | 5 | 0 | 106.2 | 17.0 | 13.0 | 14.2 | 24.1 | 19.3 | 19.3 |
| | 5 | 6 | 109.3 | 18.0 | 13.5 | 14.75 | 25.2 | 20.1 | 20.1 |
| | 6 | 0 | 111.1 | 18.5 | 14.0 | 15.25 | 26.1 | 20.8 | 20.9 |
| | 6 | 6 | 114.9 | 19.1 | 14.4 | 15.7 | 27.6 | 21.8 | 21.85 |
| | 7 | 0 | 119 1 | 20.0 | 14.9 | 16.3 | 28.7 | 22.6 | 22.8 |
| | 7 | 6 | 121.5 | 20.3 | 15.4 | 16.8 | 29.4 | 23.3 | 23.4 |
| 26 | 3 | 7 | 103.5 | 16.1 | 11.9 | 13.2 | 21.8 | 17.9 | 18.0 |
| | 4 | 3 | 105.4 | 17.1 | 12.7 | 14.1 | 23.3 | 19.0 | 19.2 |
| | 4 | 9 | 108.4 | 17.7 | 13.2 | 14.6 | 24.6 | 19.9 | 20.2 |
| | 5 | 0 | 111.5 | 18.0 | 13.5 | 14.9 | 25.0 | 20.3 | 20.6 |
| | 5 | 5 | 115.0 | 19.0 | 14.1 | 15.45 | 26.2 | 21.1 | 21.4 |
| | 6 | 0 | ..... | 19.4 | 14.6 | 16.0 | 27.4 | 22.2 | 22.4 |
| | 6 | 5 | 120.7 | 20.2 | 15.1 | 16.5 | 28.4 | 22.8 | 23.1 |
| | 7 | 0 | 124.3 | 20.8 | 15.5 | 17.05 | 29.5 | 23.6 | 23.8 |
| | 7 | 5 | 127.2 | 21.3 | 16.0 | 17.5 | 30.4 | 24.4 | 24.6 |

TABLE 5.—*Individual Data—Continued*

A. Boys—Continued

| Subject No. | Age | | Stature, Cm. | Lengths of Bones, Cm. | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Yr. | Mo. | | Humerus | Radius | Ulna | Femur | Tibia | Fibula |
| 27 | 3 | 9 | ..... | 16.0 | 11.9 | 13.0 | 22.2 | 18.0 | 17.7 |
| | 4 | 3 | ..... | 16.6 | 12.4 | 13.5 | 23.1 | 18.5 | 18.3 |
| | 4 | 6 | 104.1 | 17.0 | 12.6 | 13.7 | 23.6 | 18.9 | 18.8 |
| | 5 | 6 | 110.1 | 18.6 | 13.7 | 14.8 | 25.9 | 20.6 | 20.4 |
| | 6 | 0 | 113.8 | 19.1 | 14.1 | 15.3 | 26.7 | 21.3 | 21.0 |
| | 6 | 6 | 116.0 | 19.3 | 14.6 | 15.7 | 27.9 | 22.1 | 21.9 |
| | 7 | 0 | 119.0 | 20.0 | 14.9 | 16.1 | 28.6 | 22.8 | 22.4 |
| | 7 | 6 | 122.6 | 20.75 | 15.5 | 16.7 | 29.9 | 23.7 | 23.4 |
| | 8 | 0 | 124.9 | 21.1 | 15.8 | 17.1 | 30.8 | 24.4 | 24.0 |
| | 8 | 6 | 127.5 | 21.9 | 16.3 | 17.6 | 31.3 | 25.2 | 24.8 |
| 28 | 4 | 1 | 108.8 | 18.0 | 13.8 | 15.1 | 24.8 | 20.9 | 20.9 |
| | 5 | 0 | 116.1 | 19.4 | 15.0 | 16.4 | 27.6 | 23.0 | 23.0 |
| | 5 | 6 | 120.9 | 20.3 | 15.5 | 17.0 | 28.3 | 24.2 | 24.0 |
| | 6 | 0 | 124.2 | 21.2 | 16.1 | 17.6 | 29.6 | 25.0 | 25.0 |
| | 6 | 6 | 127.2 | 22.0 | 16.8 | 18.2 | 30.7 | 26.4 | 26.1 |
| | 7 | 0 | 132.3 | 22.9 | 17.4 | 18.9 | 32.9 | 27.5 | 27.3 |
| | 7 | 6 | 134.8 | 23.6 | 18.0 | 19.5 | 33.5 | 28.6 | 28.4 |
| | 8 | 0 | 138.6 | 24.6 | 18.6 | 20.1 | 35.1 | 29.6 | 29.3 |
| 29 | 4 | 6 | 106.7 | 18.5 | 13.5 | 14.7 | 25.3 | 20.0 | 20.05 |
| | 5 | 0 | 111.2 | 19.4 | 14.3 | 15.6 | 26.5 | 21.0 | 21.1 |
| | 5 | 6 | 114.8 | 20.2 | 14.8 | 16.2 | 28.2 | 22.1 | 22.2 |
| | 6 | 0 | 119.5 | 20.6 | 15.4 | 16.8 | 29.2 | 23.0 | 23.2 |
| | 6 | 6 | 122.9 | 21.7 | 16.0 | 17.5 | 30.5 | 23.9 | 24.1 |
| | 7 | 0 | 125.8 | 22.4 | 16.4 | 17.9 | 31.9 | 24.8 | 24.9 |
| | 7 | 6 | 129.0 | 23.1 | 16.8 | 18.4 | 32.6 | 25.5 | 25.6 |
| | 8 | 0 | 130.7 | 23.9 | 17.3 | 18.7 | 33.8 | 26.4 | 26.6 |
| | 8 | 6 | 134.8 | 24.3 | 17.65 | 19.25 | 34.6 | 26.9 | 26.9 |
| 30 | 4 | 9 | ..... | 17.9 | 12.9 | 14.6 | 24.0 | 20.3 | 20.4 |
| | 5 | 7 | 114.8 | 18.7 | 13.8 | 15.3 | 26.2 | 21.9 | 21.7 |
| | 6 | 0 | 117.1 | 19.2 | 14.1 | 15.75 | 26.9 | 22.5 | 22.5 |
| | 6 | 7 | 121.8 | 20.1 | 14.75 | 16.3 | 28.7 | 23.7 | 23.4 |
| | 7 | 0 | 123.8 | 20.7 | 15.1 | 16.7 | 29.6 | 24.6 | 24.2 |
| | 7 | 6 | 129.3 | 21.3 | 15.7 | 17.4 | 30.9 | 25.6 | 25.3 |
| | 8 | 0 | 133.3 | 22.2 | 16.1 | 17.9 | 32.0 | 26.5 | 26.3 |
| | 8 | 6 | 136.0 | 22.9 | 16.7 | 18.4 | 33.0 | 27.7 | 27.3 |
| | 9 | 0 | 139.1 | 23.7 | 17.2 | 18.9 | 34.1 | 28.5 | 28.1 |
| | 9 | 6 | 142.1 | 24.2 | 17.65 | 19.5 | 35.1 | 29.5 | 29.1 |
| 31 | 5 | 0 | 105.5 | 17.6 | 13.5 | 14.6 | 24.5 | 20.0 | 20.15 |
| | 5 | 8 | ..... | 18.7 | 14.2 | 15.5 | 26.3 | 21.2 | 21.4 |
| | 6 | 0 | 112.5 | 19.1 | 14.5 | 15.8 | 26.8 | 21.7 | 21.9 |
| | 6 | 6 | 115.3 | 19.4 | 15.05 | 16.4 | 28.2 | 22.6 | 22.8 |
| | 7 | 0 | 118.4 | 20.4 | 15.4 | 16.7 | 28.8 | 23.3 | 23.4 |
| | 7 | 5 | 121.7 | 20.7 | 15.9 | 17.1 | 29.7 | 24.1 | 24.1 |
| | 8 | 0 | 124.3 | 21.6 | 16.4 | 17.7 | 31.1 | 25.3 | 25.3 |
| | 8 | 6 | 127.1 | 21.8 | 16.9 | 18.25 | 31.9 | 26.0 | 26.05 |
| | 9 | 0 | 129.7 | 22.5 | 17.3 | 18.65 | 33.2 | 26.9 | 26.9 |
| | 9 | 6 | 132.6 | 22.8 | 17.8 | 19.1 | 33.8 | 27.5 | 27.4 |
| 32 | 5 | 0 | 109.1 | 17.9 | 12.9 | 14.2 | 24.9 | 19.8 | 19.8 |
| | 6 | 6 | 118.5 | 19.5 | 14.3 | 15.6 | 27.5 | 22.1 | 21.9 |
| | 7 | 0 | 121.0 | 20.4 | 14.8 | 16.1 | 28.6 | 22.8 | 22.8 |
| | 7 | 6 | 123.2 | 20.9 | 15.1 | 16.5 | 29.7 | 23.5 | 23.4 |
| | 8 | 0 | 124.5 | 21.5 | 15.5 | 16.8 | 30.6 | 24.3 | 24.1 |
| | 8 | 6 | 128.1 | 22.0 | 16.0 | 17.2 | 31.6 | 25.0 | 24.8 |
| | 9 | 0 | 130.5 | 22.5 | 16.2 | 17.7 | 32.4 | 25.6 | 25.5 |
| 33 | 5 | 0 | ..... | 17.8 | 13.1 | 14.7 | 25.3 | 20.0 | 20.1 |
| | 5 | 6 | ..... | 18.7 | 13.5 | 15.3 | 26.2 | 20.8 | 20.9 |
| | 6 | 3 | 120.8 | 19.5 | 14.2 | 16.1 | 27.7 | 22.0 | 22.1 |
| | 6 | 9 | 124.0 | 19.9 | 14.7 | 16.6 | 28.8 | 22.8 | 22.9 |
| | 7 | 0 | 125.0 | 20.2 | 15.0 | 16.9 | 29.2 | 23.2 | 23.3 |
| | 7 | 6 | 128.0 | 20.8 | 15.3 | 17.2 | 30.5 | 23.9 | 24.0 |
| | 8 | 0 | 131.6 | 21.6 | 15.8 | 17.7 | 31.3 | 24.7 | 24.8 |
| | 9 | 0 | 136.9 | 23.0 | 16.6 | 18.7 | 33.5 | 26.3 | 26.3 |
| | 9 | 6 | 139.1 | 23.3 | 16.9 | 19.2 | 34.0 | 27.0 | 27.0 |
| 34 | 5 | 3 | 111.0 | 18.2 | 13.2 | 14.4 | 25.2 | 20.7 | 20.35 |
| | 5 | 10 | 114.7 | 19.1 | 13.8 | 15.1 | 26.5 | 21.7 | 21.4 |
| | 6 | 3 | 117.1 | 19.6 | 14.1 | 15.45 | 27.5 | 22.4 | 22.0 |
| | 6 | 6 | 119.5 | 20.0 | 14.4 | 15.7 | 28.3 | 22.8 | 22.4 |
| | 9 | 0 | 133.1 | 23.1 | 16.75 | 18.1 | 32.9 | 26.9 | 26.2 |
| 35 | 5 | 3 | 109.4 | 17.0 | 12.4 | 13.8 | 23.9 | 18.8 | 18.75 |
| | 6 | 3 | 115.7 | 18.4 | 13.4 | 14.8 | 26.2 | 20.5 | 20.2 |
| | 7 | 0 | 121.4 | 19.2 | 14.2 | 15.6 | 27.5 | 21.6 | 21.4 |
| | 7 | 6 | 124.0 | 19.7 | 14.5 | 16.0 | 29.1 | 22.5 | 22.2 |
| | 7 | 11 | 126.2 | 20.4 | 14.9 | 16.35 | 29.5 | 23.0 | 22.8 |
| | 8 | 6 | 130.6 | 21.4 | 15.5 | 17.0 | 31.0 | 24.1 | 23.9 |
| | 9 | 0 | 133.4 | 21.6 | 15.9 | 17.5 | 32.3 | 24.8 | 24.6 |
| | 9 | 6 | 137.3 | 22.3 | 16.3 | 17.9 | 32.6 | 25.5 | 25.2 |

TABLE 5.—*Individual Data—Continued*

A. Boys—Continued

| Subject No. | Age | | Stature, Cm. | Lengths of Bones, Cm. | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Yr. | Mo. | | Humerus | Radius | Ulna | Femur | Tibia | Fibula |
| 36 | 5 | 9 | ..... | 18.7 | 14.0 | 15.3 | 26.8 | 20.9 | 20.85 |
| | 6 | 6 | 119.1 | 19.7 | 14.55 | 15.9 | 28.2 | 22.0 | 22.0 |
| | 7 | 0 | 122.3 | 20.2 | 15.1 | 16.5 | 29.3 | 22.8 | 22.8 |
| | 7 | 6 | 124.6 | 20.8 | 15.6 | 16.9 | 30.3 | 23.6 | 23.45 |
| | 8 | 0 | 127.2 | 21.4 | 15.95 | 17.3 | 30.8 | 24.2 | 24.1 |
| | 8 | 6 | 131.0 | 22.0 | 16.4 | 17.8 | 32.4 | 25.3 | 25.1 |
| | 9 | 0 | 133.1 | 22.8 | 16.9 | 18.2 | 33.2 | 25.8 | 25.8 |
| | 9 | 6 | 136.1 | 23.3 | 17.2 | 18.6 | 34.6 | 26.5 | 26.6 |
| | 10 | 0 | 137.9 | 23.8 | 17.6 | 19.0 | 35.2 | 27.2 | 27.2 |
| 37 | 5 | 9 | 115.6 | 18.9 | 14.3 | 15.6 | 26.6 | 21.7 | 21.4 |
| | 6 | 9 | 121.4 | 20.2 | 15.2 | 16.6 | 28.5 | 23.4 | 22.9 |
| | 7 | 3 | 124.7 | 20.8 | 15.7 | 17.1 | 29.6 | 24.3 | 23.9 |
| | 8 | 0 | 129.6 | 21.5 | 16.4 | 17.8 | 30.8 | 25.6 | 25.1 |
| | 9 | 0 | 134.9 | 22.6 | 17.3 | 18.8 | 32.8 | 27.1 | 26.6 |
| | 9 | 7 | 138.4 | 23.5 | 17.8 | 19.2 | 34.0 | 28.0 | 27.5 |
| 38 | 5 | 10 | 120.4 | 20.3 | 14.2 | 15.4 | 28.7 | 22.6 | 22.6 |
| | 6 | 7 | ..... | 21.4 | 14.9 | 16.1 | 30.6 | 23.6 | 23.7 |
| | 6 | 9 | 126.3 | 21.5 | 15.1 | 16.3 | 31.0 | 24.0 | 24.0 |
| | 7 | 4 | 129.9 | 22.2 | 15.6 | 16.8 | 32.0 | 24.9 | 24.9 |
| | 7 | 6 | 131.4 | 22.3 | 15.75 | 17.0 | 32.3 | 25.3 | 25.3 |
| | 8 | 0 | 133.9 | 23.4 | 16.3 | 17.5 | 33.7 | 26.3 | 26.2 |
| | 8 | 6 | 137.2 | 23.8 | 16.8 | 17.9 | 34.4 | 27.0 | 26.9 |
| | 9 | 0 | 139.3 | 24.4 | 17.2 | 18.4 | 35.5 | 27.9 | 27.8 |
| | 9 | 6 | 142.5 | 25.0 | 17.6 | 18.8 | 36.6 | 28.5 | 28.4 |
| | 10 | 0 | 144.9 | 25.5 | 18.0 | 19.3 | 37.6 | 29.2 | 29.1 |
| 39 | 5 | 10 | 117.2 | 19.7 | 15.2 | 16.2 | 27.5 | 22.35 | 22.5 |
| | 6 | 9 | 123.2 | 20.3 | 16.2 | 17.3 | 29.8 | 24.2 | 24.2 |
| | 7 | 6 | 128.5 | 21.7 | 17.0 | 18.0 | 31.2 | 25.3 | 25.4 |
| | 8 | 0 | 132.1 | 22.7 | 17.5 | 18.5 | 32.5 | 26.3 | 26.3 |
| | 8 | 6 | 134.0 | 23.1 | 17.9 | 19.05 | 33.2 | 27.0 | 26.9 |
| | 9 | 0 | 138.2 | 23.7 | 18.5 | 19.8 | 34.6 | 27.9 | 27.9 |
| | 9 | 6 | 140.0 | 24.9 | 18.9 | 20.3 | 35.3 | 28.7 | 28.6 |
| 40 | 5 | 11 | 118.1 | 19.0 | 14.5 | 15.5 | 27.6 | 22.1 | 22.0 |
| | 6 | 10 | 126.1 | 20.4 | 15.5 | 16.6 | 29.4 | 23.5 | 23.4 |
| | 7 | 6 | 130.5 | 21.2 | 16.3 | 17.3 | 31.2 | 24.7 | 24.6 |
| | 8 | 1 | 133.2 | 22.4 | 16.75 | 17.8 | 31.9 | 25.5 | 25.3 |
| | 8 | 7 | 136.2 | 22.5 | 17.25 | 18.4 | 33.2 | 26.3 | 26.2 |
| | 9 | 0 | 139.1 | 23.3 | 17.5 | 18.6 | 33.9 | 26.9 | 26.75 |
| | 9 | 6 | 141.6 | 23.8 | 17.9 | 19.1 | 34.6 | 27.7 | 27.5 |
| | 10 | 0 | 144.4 | 24.5 | 18.3 | 19.5 | 35.7 | 28.2 | 28.0 |
| 41 | 6 | 0 | 116.2 | 18.9 | 14.2 | 15.5 | 27.3 | 21.8 | 21.8 |
| | 8 | 0 | 129.1 | 21.5 | 16.3 | 17.5 | 31.9 | 25.5 | 25.4 |
| | 8 | 6 | 132.8 | 22.1 | 16.7 | 18.0 | 32.8 | 26.1 | 26.2 |
| | 9 | 0 | 135.4 | 22.7 | 17.1 | 18.3 | 33.7 | 27.0 | 27.0 |
| | 9 | 6 | 137.5 | 23.0 | 17.5 | 18.8 | 34.9 | 27.7 | 27.8 |
| | 10 | 0 | 141.8 | 23.5 | 17.9 | 19.2 | 35.7 | 28.5 | 28.6 |
| 42 | 6 | 0 | 117.9 | 20.2 | 14.7 | 16.3 | 29.5 | 22.7 | 22.4 |
| | 6 | 9 | 122.4 | 21.0 | 15.4 | 17.0 | 31.0 | 23.9 | 23.5 |
| | 8 | 6 | 131.1 | 23.1 | 16.95 | 18.8 | 33.7 | 26.5 | 25.95 |
| | 9 | 6 | 137.0 | 24.1 | 17.9 | 19.7 | 35.9 | 28.2 | 27.4 |
| | 10 | 0 | 140.7 | 24.7 | 18.4 | 20.2 | 37.1 | 29.2 | 28.4 |
| 43 | 6 | 4 | ..... | 20.6 | 15.0 | 16.3 | 28.4 | 23.2 | 22.8 |
| | 7 | 7 | 129.9 | 22.5 | 16.3 | 17.6 | 31.3 | 25.3 | 25.0 |
| | 8 | 0 | 131.6 | 22.8 | 16.8 | 18.15 | 32.1 | 26.0 | 25.7 |
| | 8 | 6 | 135.1 | 23.4 | 17.2 | 18.55 | 33.0 | 26.9 | 26.5 |
| | 9 | 0 | ..... | 24.0 | 17.7 | 19.2 | 34.2 | 27.7 | 27.3 |
| | 9 | 6 | 141.1 | 24.9 | 18.15 | 19.55 | 35.2 | 28.5 | 28.1 |
| | 10 | 0 | 144.2 | 25.3 | 18.6 | 20.0 | 35.9 | 29.4 | 28.9 |
| | 10 | 6 | 146.8 | 26.0 | 18.9 | 20.5 | 36.6 | 30.1 | 29.7 |
| 44 | 6 | 5 | 121.3 | 20.7 | 15.2 | 16.5 | 28.8 | 24.1 | 24.0 |
| | 7 | 1 | 125.0 | 21.5 | 15.9 | 17.2 | 30.0 | 25.3 | 25.2 |
| | 8 | 0 | 132.0 | 23.3 | 17.1 | 18.4 | 32.3 | 27.3 | 27.05 |
| | 8 | 6 | 133.3 | 23.8 | 17.4 | 18.85 | 33.4 | 28.0 | 27.7 |
| | 9 | 0 | 137.0 | 24.7 | 18.0 | 19.5 | 34.8 | 29.0 | 28.75 |
| | 9 | 6 | 138.9 | 25.3 | 18.4 | 20.0 | 35.6 | 29.8 | 29.5 |
| | 10 | 0 | 142.6 | 25.5 | 19.0 | 20.4 | 36.5 | 30.5 | 30.4 |
| 45 | 6 | 9 | 120.8 | 19.7 | 14.8 | 16.3 | 28.3 | 23.2 | 23.0 |
| 46 | 7 | 0 | ..... | 19.8 | 14.9 | 16.3 | 27.3 | 23.5 | 23.1 |
| | 8 | 6 | 130.8 | 21.6 | 16.2 | 18.0 | 30.5 | 26.2 | 25.7 |
| | 9 | 0 | 131.7 | 22.1 | 16.7 | 18.35 | 31.3 | 27.0 | 26.5 |
| | 9 | 6 | ..... | 22.7 | 17.05 | 18.8 | 32.2 | 27.8 | 27.3 |
| | 10 | 6 | 140.3 | 23.9 | 17.9 | 19.5 | 34.1 | 29.5 | 28.9 |
| | 11 | 0 | 143.2 | 24.4 | 18.3 | 19.9 | 34.8 | 30.4 | 29.7 |
| 47 | 7 | 2 | ..... | 21.7 | 16.6 | 17.7 | 30.4 | 25.2 | 25.3 |
| | 9 | 5 | 139.0 | 24.4 | 18.8 | 19.9 | 34.8 | 29.1 | 29.0 |
| | 10 | 0 | 143.0 | 25.1 | 19.5 | 20.7 | 36.1 | 30.2 | 30.1 |
| | 10 | 6 | 146.5 | 25.8 | 19.9 | 21.3 | 37.3 | 31.1 | 31.0 |
| | 11 | 0 | 149.2 | 26.4 | 20.4 | 21.7 | 38.4 | 31.9 | 31.7 |
| | 11 | 6 | 152.3 | 27.1 | 20.9 | 22.3 | 39.2 | 33.0 | 32.7 |

TABLE 5.—*Individual Data—Continued*

A. Boys—Continued

| Subject No. | Age | | Stature, Cm. | Lengths of Bones, Cm. | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Yr. | Mo. | | Humerus | Radius | Ulna | Femur | Tibia | Fibula |
| 48 | 7 | 3 | ..... | 19.8 | 14.9 | 16.15 | 27.8 | 22.6 | 22.2 |
| | 7 | 9 | 125.0 | 20.2 | 15.4 | 16.7 | 28.8 | 23.4 | 22.9 |
| | 8 | 6 | 128.6 | 21.25 | 16.1 | 17.4 | 30.5 | 24.6 | 24.1 |
| | 9 | 0 | 132.1 | 21.8 | 16.6 | 17.9 | 31.5 | 25.4 | 25.0 |
| | 9 | 6 | ..... | 22.4 | 17.0 | 18.3 | 31.9 | 26.3 | 25.8 |
| | 10 | 0 | 136.6 | 22.7 | 17.5 | 18.75 | 33.4 | 27.0 | 26.4 |
| | 10 | 6 | 139.9 | 23.4 | 17.9 | 19.3 | 34.3 | 27.8 | 27.1 |
| | 11 | 0 | 142.4 | 23.7 | 18.35 | 19.6 | 34.6 | 28.4 | 27.7 |
| 49 | 7 | 6 | 122.0 | 20.6 | 15.4 | 16.8 | 28.2 | 23.4 | 23.1 |
| | 9 | 6 | 134.3 | 23.1 | 17.6 | 19.1 | 32.3 | 27.0 | 26.4 |
| | 10 | 0 | ..... | 24.3 | 18.1 | 19.6 | 33.1 | 27.8 | 27.1 |
| | 10 | 6 | 139.5 | 24.9 | 18.55 | 20.2 | 34.2 | 28.6 | 27.8 |
| | 11 | 0 | 141.4 | 25.1 | 18.8 | 20.7 | 35.0 | 29.3 | 28.5 |
| | 11 | 6 | 144.2 | 25.8 | 19.3 | 21.5 | 36.0 | 29.9 | 29.1 |
| 50 | 7 | 6 | ..... | 20.0 | 15.3 | 16.5 | 28.2 | 22.0 | 22.0 |
| | 9 | 6 | 128.1 | 22.2 | 16.9 | 18.2 | 31.5 | 24.6 | 24.8 |
| | 10 | 6 | 133.7 | 23.6 | 17.8 | 18.9 | 33.3 | 26.0 | 26.15 |
| | 11 | 6 | 138.3 | 24.3 | 18.4 | 19.8 | 34.8 | 27.0 | 27.1 |
| 51 | 7 | 9 | 127.8 | 21.9 | 16.4 | 17.9 | 30.9 | 25.5 | 25.4 |
| | 8 | 3 | 130.4 | 22.8 | 16.9 | 18.5 | 31.8 | 26.4 | 26.3 |
| | 9 | 0 | 134.2 | 23.2 | 17.4 | 19.0 | 33.2 | 27.5 | 27.3 |
| | 9 | 6 | 137.0 | 24.3 | 18.0 | 19.6 | 34.2 | 28.4 | 28.1 |
| | 10 | 0 | 138.6 | 24.8 | 18.5 | 20.2 | 34.7 | 29.1 | 28.9 |
| | 10 | 6 | 141.6 | 25.2 | 18.8 | 20.4 | 35.7 | 29.9 | 29.6 |
| | 11 | 0 | 143.8 | 25.4 | 19.2 | 21.0 | 36.3 | 30.6 | 30.2 |
| | 11 | 6 | 146.0 | 26.2 | 19.6 | 21.0 | 37.1 | 31.4 | 30.9 |
| 52 | 7 | 9 | ..... | 23.2 | 17.9 | 19.4 | 31.9 | 27.8 | 27.5 |
| | 10 | 0 | 144.6 | 25.6 | 19.9 | 21.4 | 35.7 | 31.1 | 30.6 |
| 53 | 8 | 9 | ..... | 22.6 | 16.6 | 18.1 | 31.7 | 25.7 | 25.4 |
| | 10 | 0 | 138.1 | 24.2 | 17.6 | 19.1 | 34.1 | 27.5 | 27.2 |
| | 10 | 6 | 140.5 | 24.5 | 18.0 | 19.6 | 34.9 | 28.2 | 27.7 |
| | 11 | 0 | 142.5 | 25.4 | 18.4 | 19.9 | 35.8 | 28.8 | 28.3 |
| | 11 | 6 | 145.4 | 25.9 | 18.8 | 20.4 | 37.0 | 29.7 | 29.15 |
| | 12 | 0 | 148.2 | .... | 19.1 | 20.8 | 37.9 | .... | .... |
| 54 | 8 | 10 | 137.0 | 23.5 | 17.45 | 19.0 | 34.1 | 28.3 | 28.5 |
| | 9 | 6 | 141.7 | 24.5 | 18.1 | 19.7 | 35.6 | 29.6 | 29.7 |
| | 10 | 0 | 144.8 | 25.2 | 18.5 | 20.1 | 36.3 | 30.5 | 30.6 |
| | 10 | 6 | 147.3 | 25.4 | 18.9 | 20.6 | 37.6 | 31.5 | 31.7 |
| | 11 | 0 | 149.5 | 26.1 | 19.5 | 21.1 | 38.7 | 32.3 | 32.4 |
| | 11 | 6 | 152.5 | 26.8 | 20.0 | 21.6 | 39.2 | 33.3 | 33.2 |
| | 12 | 0 | 156.2 | 27.5 | 20.5 | 22.1 | 40.5 | 34.2 | 34.15 |
| 55 | 9 | 0 | 131.5 | 23.7 | 16.5 | 17.9 | 32.8 | 26.0 | 25.6 |
| 56 | 9 | 6 | 140.4 | 22.2 | 17.0 | 18.3 | 33.3 | 26.7 | 26.75 |
| | 10 | 0 | 143.2 | 23.0 | 17.45 | 18.8 | 34.0 | 27.7 | 27.6 |
| | 10 | 6 | 146.4 | 23.8 | 17.95 | 19.4 | 35.7 | 28.6 | 28.5 |
| | 11 | 0 | 149.9 | 25.1 | 18.3 | 19.7 | 36.3 | 29.3 | 29.2 |
| | 11 | 6 | 153.7 | 25.4 | 18.8 | 20.4 | 37.5 | 30.4 | 30.2 |
| | 12 | 0 | 158.8 | 26.4 | 19.45 | 21.3 | 38.9 | 31.5 | 31.2 |
| 57 | 10 | 3 | 145.8 | 26.2 | 19.8 | 21.6 | 38.0 | 32.2 | 31.0 |
| 58 | 10 | 4 | 136.0 | 23.4 | 17.9 | 19.3 | 33.8 | 28.6 | 27.5 |
| | 11 | 4 | 140.3 | 24.3 | 18.8 | 20.2 | 35.4 | 29.9 | 28.7 |
| | 12 | 0 | 143.7 | 25.0 | 19.4 | 20.9 | 36.9 | 31.0 | 29.8 |
| 59 | 10 | 6 | 146.9 | 25.5 | 19.9 | 21.6 | 37.5 | 30.0 | 30.1 |
| | 11 | 0 | 149.7 | 25.9 | 20.3 | 22.1 | 38.1 | 30.8 | 30.8 |
| | 11 | 6 | ..... | 26.7 | 20.8 | 22.8 | 38.85 | 31.7 | 31.7 |
| | 12 | 0 | 156.3 | 27.4 | 21.3 | 23.1 | 40.5 | 32.6 | 32.6 |
| 60 | 11 | 6 | 137.2 | 22.4 | 18.0 | 19.6 | 33.0 | 27.8 | 27.3 |
| | 12 | 0 | 140.0 | 22.8 | 18.35 | 20.05 | 33.7 | 28.4 | 27.95 |
| 61 | 11 | 6 | 143.3 | 24.7 | 18.3 | 20.3 | 34.5 | 28.7 | 28.4 |
| | 12 | 0 | 145.7 | 25.0 | 18.8 | 20.8 | 35.2 | 29.5 | 29.2 |
| | | | | | B. Girls | | | | |
| 1 | 0 | 2 | 51.8 | 6.4 | 5.25 | 5.8 | 8.1 | 6.6 | 6.15 |
| | 0 | 6 | 60.5 | 8.1 | 6.05 | 6.8 | 10.4 | 8.2 | 7.6 |
| 2 | 0 | 2 | 53.1 | 6.9 | 5.65 | 6.45 | 7.8 | 6.5 | 6.1 |
| | 0 | 4 | 59.2 | 7.8 | 6.25 | 7.0 | 9.5 | 7.7 | 7.5 |
| | 0 | 6 | 66.0 | 8.95 | 6.85 | 7.7 | 11.0 | 8.7 | 8.5 |
| 3 | 0 | 2 | 54.0 | 6.8 | 5.2 | 6.1 | 8.4 | 6.5 | 6.2 |
| | 0 | 4 | 59.7 | 7.5 | 5.7 | 6.55 | 9.5 | 7.3 | 6.8 |
| | 0 | 6 | 62.7 | 8.45 | 6.2 | 7.1 | 10.7 | 8.3 | 7.9 |
| 4 | 0 | 2 | 55.4 | 7.05 | 5.9 | 6.6 | 9.15 | 7.45 | 7.15 |

TABLE 5.—*Individual Data—Continued*

B. Girls—Continued

| Subject No. | Age Yr. | Age Mo. | Stature, Cm. | Lengths of Bones, Cm. Humerus | Radius | Ulna | Femur | Tibia | Fibula |
|---|---|---|---|---|---|---|---|---|---|
| 5 | 0 | 3 | 62.0 | 7.9 | 6.4 | 7.2 | 9.45 | 7.5 | 7.3 |
| | 1 | 0 | 79.7 | 11.1 | 8.2 | 9.1 | 13.9 | 11.4 | 11.0 |
| | 1 | 6 | 83.4 | 12.3 | 9.1 | 10.2 | 15.8 | 12.8 | 12.5 |
| | 2 | 0 | 87.2 | 13.55 | 9.85 | 11.0 | 17.3 | 14.1 | 13.9 |
| 6 | 0 | 3 | 59.0 | 6.8 | 6.1 | 6.9 | 9.7 | 7.0 | 6.9 |
| | 0 | 7 | 66.5 | 8.9 | 6.9 | 7.65 | 11.2 | 8.2 | 8.1 |
| | 1 | 0 | 69.4 | 9.7 | 7.8 | 8.7 | 13.4 | 10.5 | 10.5 |
| | 1 | 6 | 75.5 | 11.1 | 8.5 | 9.5 | 15.1 | 12.0 | 12.0 |
| 7 | 0 | 6 | 68.2 | 9.1 | 6.9 | 7.6 | 11.55 | 9.2 | 8.8 |
| | 1 | 0 | 77.1 | 10.6 | 8.2 | 9.1 | 14.6 | 11.7 | 11.2 |
| | 1 | 6 | 84.4 | 12.2 | 9.1 | 10.2 | 16.7 | 13.1 | 12.75 |
| | 2 | 0 | 89.9 | 13.6 | 9.8 | 10.75 | 18.3 | 14.4 | 13.95 |
| 8 | 0 | 6 | 68.2 | 9.0 | 6.95 | 7.7 | 11.2 | 8.8 | 8.8 |
| | 1 | 0 | 74.4 | 10.7 | 8.05 | 9.0 | 13.5 | 10.8 | 10.5 |
| | 1 | 6 | 81.3 | 12.3 | 9.0 | 10.1 | 15.6 | 12.7 | 12.45 |
| | 2 | 0 | 86.7 | 13.1 | 9.6 | 10.6 | 16.8 | 13.7 | 13.45 |
| 9 | 0 | 6 | 67.0 | 9.55 | 7.3 | 7.95 | 12.0 | 9.7 | 9.4 |
| | 1 | 0 | 74.3 | 11.0 | 8.5 | 9.4 | 14.4 | 11.6 | 11.3 |
| | 1 | 6 | 81.9 | 12.7 | 9.3 | 10.5 | 16.5 | 13.3 | 13.2 |
| 10 | 0 | 6 | 65.8 | 8.9 | 6.7 | 7.4 | 11.2 | 9.0 | 8.55 |
| | 1 | 0 | 74.6 | 10.5 | 7.8 | 8.8 | 13.9 | 11.0 | 10.6 |
| | 1 | 6 | 82.2 | 11.95 | 8.85 | 10.0 | 16.3 | 12.7 | 12.4 |
| 11 | 0 | 6 | 64.1 | 8.8 | 6.5 | 7.3 | 11.0 | 8.7 | 8.3 |
| | 1 | 0 | 72.0 | 10.5 | 7.75 | 8.65 | 13.35 | 10.8 | 10.4 |
| | 1 | 6 | 79.0 | 11.6 | 8.65 | 9.6 | 15.1 | 12.5 | 12.1 |
| | 2 | 0 | 85.9 | 12.95 | 9.4 | 10.4 | 16.8 | 14.0 | 13.4 |
| 12 | 0 | 6 | 65.7 | 9.0 | 7.2 | 7.85 | 11.3 | 9.3 | 8.6 |
| | 1 | 0 | 76.2 | 10.85 | 8.4 | 9.35 | 13.7 | 11.4 | 10.9 |
| 13 | 0 | 6 | 65.3 | 9.0 | 6.8 | 7.5 | 10.8 | 9.0 | 8.6 |
| | 1 | 0 | 72.7 | 10.6 | 7.8 | 8.8 | 13.15 | 10.8 | 10.5 |
| | 1 | 6 | 77.9 | 11.2 | 8.5 | 9.6 | 14.9 | 12.1 | 11.9 |
| | 2 | 0 | 81.6 | 12.4 | 9.0 | 10.15 | 16.4 | 13.4 | 13.2 |
| | 2 | 6 | 85.4 | 13.1 | 9.7 | 10.9 | 17.7 | 14.3 | 14.15 |
| | 3 | 0 | 89.2 | 13.9 | 10.2 | 11.45 | 19.0 | 15.2 | 15.15 |
| 14 | 0 | 9 | 71.0 | 10.1 | 7.8 | 8.8 | 12.4 | 10.0 | 9.95 |
| | 1 | 6 | 80.7 | 11.8 | 9.1 | 10.2 | 15.6 | 12.4 | 12.6 |
| | 2 | 0 | 85.0 | 13.2 | 9.7 | 11.0 | 17.2 | 13.5 | 13.9 |
| 15 | 0 | 9 | 72.7 | 9.8 | 7.7 | 9.0 | 12.7 | 10.5 | 10.3 |
| | 1 | 0 | 77.1 | 10.5 | 8.25 | 9.7 | 13.9 | 11.0 | 10.9 |
| | 1 | 6 | 82.5 | 11.8 | 9.05 | 10.55 | 15.6 | 12.4 | 12.6 |
| | 2 | 0 | 88.0 | 12.9 | 9.8 | 11.4 | 17.3 | 14.2 | 14.2 |
| | 2 | 6 | 93.0 | 13.8 | 10.5 | 12.0 | 19.0 | 15.2 | 15.25 |
| | 3 | 0 | 96.7 | 14.6 | 11.0 | 12.5 | 19.7 | 16.2 | 16.4 |
| 16 | 0 | 9 | 68.8 | 9.5 | 7.4 | 8.4 | 12.0 | 9.4 | 8.9 |
| | 1 | 0 | 71.6 | 10.25 | 7.8 | 8.8 | 12.6 | 10.0 | 9.5 |
| | 1 | 6 | 77.2 | 11.6 | 8.6 | 9.7 | 14.5 | 11.5 | 11.15 |
| | 2 | 0 | 82.7 | 12.6 | 9.3 | 10.5 | 16.1 | 12.8 | 12.5 |
| | 2 | 6 | 86.7 | 13.8 | 9.9 | 11.0 | 17.75 | 14.2 | 13.9 |
| | 3 | 0 | 91.2 | 14.3 | 10.45 | 11.75 | 19.0 | 15.1 | 14.85 |
| 17 | 0 | 9 | 70.4 | 9.6 | 7.1 | 8.2 | 12.2 | 10.2 | 9.5 |
| | 1 | 0 | 75.3 | 10.4 | 7.7 | 8.8 | 13.5 | 10.9 | 10.3 |
| | 1 | 6 | 82.1 | 11.6 | 8.6 | 10.0 | 15.4 | 13.0 | 12.4 |
| | 2 | 0 | 86.7 | 12.4 | 9.2 | 10.7 | 16.8 | 14.4 | 13.7 |
| | 2 | 6 | 89.7 | 13.5 | 9.75 | 11.2 | 18.3 | 15.5 | 14.9 |
| | 3 | 0 | 96.1 | 14.1 | 10.45 | 11.8 | 19.8 | 16.7 | 16.3 |
| | 3 | 6 | 98.4 | 14.85 | 11.05 | 12.5 | 20.8 | 17.6 | 17.2 |
| 18 | 0 | 9 | 65.9 | 9.0 | 6.9 | 7.95 | 11.5 | 9.4 | 8.9 |
| | 1 | 6 | 75.3 | 10.9 | 8.1 | 9.3 | 14.3 | 11.4 | 11.2 |
| | 2 | 0 | 79.2 | 11.5 | 8.5 | 9.8 | 15.8 | 12.4 | 12.1 |
| | 2 | 6 | 82.9 | 12.7 | 9.3 | 10.7 | 17.2 | 13.8 | 13.7 |
| | 3 | 0 | 86.5 | 13.6 | 9.8 | 11.2 | 18.4 | 14.6 | 14.6 |
| | 3 | 6 | 90.0 | 14.3 | 10.35 | 11.8 | 19.4 | 15.5 | 15.55 |
| | 4 | 0 | 94.0 | 15.0 | 10.85 | 12.4 | 20.5 | 16.5 | 16.55 |
| 19 | 1 | 0 | 76.0 | 10.4 | 7.75 | 8.7 | 13.4 | 10.5 | 10.4 |
| | 1 | 6 | 84.5 | 11.7 | 8.55 | 9.8 | 15.4 | 12.4 | 12.2 |
| | 2 | 0 | 88.2 | 12.9 | 9.4 | 10.8 | 17.2 | 14.05 | 13.9 |
| | 2 | 6 | 93.5 | 13.8 | 10.0 | 11.5 | 19.0 | 15.2 | 15.05 |
| | 3 | 0 | 98.7 | 14.5 | 10.6 | 12.1 | 20.3 | 16.4 | 16.3 |
| | 3 | 6 | 102.5 | 15.5 | 11.2 | 12.8 | 21.8 | 17.55 | 17.4 |
| 20 | 1 | 0 | 71.0 | 10.05 | 7.6 | 8.5 | 12.8 | 10.4 | 9.95 |
| | 1 | 6 | 79.1 | 11.2 | 8.4 | 9.4 | 14.7 | 11.9 | 11.6 |
| | 2 | 0 | 83.1 | 12.1 | 9.05 | 10.1 | 16.2 | 13.2 | 13.0 |
| | 2 | 6 | 86.6 | 13.0 | 9.7 | 10.7 | 17.6 | 14.5 | 14.35 |
| | 3 | 0 | 88.1 | 13.8 | 10.3 | 11.4 | 18.6 | 15.4 | 15.4 |
| | 3 | 6 | 91.6 | 14.4 | 10.8 | 11.9 | 19.9 | 16.45 | 16.4 |
| | 4 | 0 | 96.1 | 15.2 | 11.3 | 12.5 | 21.15 | 17.6 | 17.6 |
| | 4 | 6 | 99.0 | 16.0 | 11.8 | 12.9 | 22.3 | 18.5 | 18.5 |

TABLE 5.—*Individual Data—Continued*

B. Girls—Continued

| Subject No. | Age Yr. | Age Mo. | Stature, Cm. | Lengths of Bones, Cm. Humerus | Radius | Ulna | Femur | Tibia | Fibula |
|---|---|---|---|---|---|---|---|---|---|
| 21 | 1 | 3 | 81.3 | 11.2 | 8.05 | 9.2 | 14.6 | 11.6 | 11.0 |
| | 2 | 0 | 89.3 | 12.8 | 9.15 | 10.4 | 17.2 | 13.7 | 13.1 |
| | 2 | 6 | 93.8 | 13.6 | 9.8 | 11.1 | 18.6 | 15.0 | 14.3 |
| | 3 | 0 | 96.0 | 14.3 | 10.3 | 11.8 | 20.1 | 16.1 | 15.4 |
| | 3 | 6 | 100.4 | 15.3 | 11.05 | 12.4 | 21.0 | 17.1 | 16.35 |
| | 4 | 0 | 104.1 | 16.1 | 11.6 | 13.1 | 22.8 | 18.2 | 17.55 |
| | 4 | 6 | 107.3 | 16.5 | 12.0 | 13.5 | 23.9 | 19.0 | 18.3 |
| 22 | 1 | 6 | 84.0 | 11.8 | 8.8 | 9.95 | 16.1 | 12.7 | 12.5 |
| | 2 | 0 | 91.3 | 13.6 | 9.7 | 10.9 | 17.9 | 14.5 | 14.3 |
| | 2 | 6 | 95.6 | 14.7 | 10.45 | 11.8 | 19.4 | 15.4 | 15.4 |
| | 3 | 0 | 97.8 | 15.7 | 11.2 | 12.6 | 21.0 | 16.6 | 16.7 |
| | 3 | 6 | 101.1 | 16.4 | 11.7 | 13.1 | 22.5 | 17.5 | 17.6 |
| | 4 | 0 | 104.9 | 17.0 | 12.3 | 13.8 | 23.5 | 18.5 | 18.7 |
| | 4 | 6 | 108.0 | 18.0 | 12.9 | 14.4 | 24.8 | 19.5 | 19.6 |
| 23 | 1 | 6 | 83.3 | 12.4 | 9.25 | 10.4 | 16.6 | 13.1 | 12.7 |
| | 2 | 6 | 94.3 | 14.8 | 10.7 | 12.0 | 19.9 | 15.6 | 15.4 |
| | 3 | 0 | 100.4 | 15.6 | 11.3 | 12.6 | 21.4 | 17.0 | 16.7 |
| | 3 | 6 | 102.5 | 16.7 | 12.05 | 13.4 | 22.9 | 18.2 | 18.1 |
| | 4 | 0 | ..... | 17.5 | 12.6 | 14.0 | 24.1 | 19.2 | 19.1 |
| | 4 | 6 | 110.4 | 18.2 | 13.2 | 14.6 | 25.7 | 20.3 | 20.2 |
| 24 | 2 | 0 | 87.1 | 12.8 | 10.0 | 11.0 | 17.1 | 13.8 | 13.6 |
| | 2 | 6 | 93.4 | 14.1 | 10.75 | 11.85 | 18.8 | 15.0 | 14.9 |
| | 3 | 0 | 96.7 | 15.2 | 11.55 | 12.7 | 20.3 | 16.2 | 16.1 |
| | 3 | 6 | 100.7 | 16.0 | 12.1 | 13.2 | 21.7 | 17.3 | 17.1 |
| | 4 | 0 | 105.0 | 16.5 | 12.85 | 14.1 | 23.0 | 18.4 | 18.2 |
| | 4 | 6 | 109.4 | 17.6 | 13.4 | 14.7 | 24.6 | 19.4 | 19.1 |
| 25 | 2 | 6 | 94.5 | 13.2 | 10.55 | 11.7 | 19.0 | 15.5 | 15.2 |
| | 3 | 6 | 101.4 | 15.8 | 11.7 | 13.0 | 21.5 | 17.6 | 17.4 |
| | 4 | 0 | ..... | 16.4 | 12.2 | 13.5 | 22.7 | 18.5 | 18.2 |
| | 4 | 6 | 108.1 | 17.3 | 12.9 | 14.3 | 23.9 | 19.4 | 19.4 |
| | 5 | 1 | 111.9 | 18.1 | 13.6 | 15.1 | 25.3 | 20.8 | 20.6 |
| | 5 | 6 | 116.4 | 18.8 | 14.1 | 15.55 | 26.5 | 21.7 | 21.45 |
| 26 | 2 | 6 | 86.0 | 12.9 | 8.7 | 9.8 | 17.0 | 13.7 | 13.8 |
| | 3 | 0 | 90.8 | 13.35 | 9.8 | 11.1 | 17.8 | 14.0 | 14.1 |
| | 3 | 6 | 94.5 | 14.1 | 10.4 | 11.7 | 19.1 | 15.0 | 15.1 |
| | 4 | 0 | 96.8 | 14.8 | 10.85 | 12.25 | 20.1 | 15.7 | 15.8 |
| | 4 | 6 | 100.1 | 15.5 | 11.3 | 12.9 | 21.2 | 16.7 | 16.65 |
| | 5 | 0 | 103.7 | 16.2 | 11.9 | 13.5 | 22.4 | 17.5 | 17.4 |
| | 5 | 6 | 107.6 | 16.9 | 12.3 | 13.9 | 23.3 | 18.3 | 18.3 |
| 27 | 2 | 9 | ..... | 15.6 | 11.7 | 12.8 | 20.4 | 17.1 | 16.7 |
| | 3 | 9 | 106.5 | 17.2 | 12.8 | 14.1 | 22.8 | 19.2 | 18.9 |
| | 4 | 3 | 107.3 | 17.9 | 13.4 | 14.7 | 24.0 | 20.3 | 19.9 |
| | 4 | 7 | 110.2 | 18.5 | 13.8 | 15.1 | 25.0 | 21.0 | 20.6 |
| | 5 | 0 | 113.7 | 19.2 | 14.2 | 15.6 | 25.8 | 21.9 | 21.5 |
| | 5 | 6 | ..... | 19.9 | 14.8 | 16.2 | 27.1 | 22.7 | 22.4 |
| | 6 | 0 | 120.2 | 20.6 | 15.4 | 16.6 | 28.6 | 23.8 | 23.5 |
| | 6 | 6 | 124.8 | 21.3 | 15.8 | 17.3 | 29.7 | 24.75 | 24.3 |
| | 7 | 0 | 126.6 | 22.0 | 16.3 | 17.6 | 30.5 | 25.6 | 25.1 |
| 28 | 3 | 0 | ..... | 15.6 | 11.3 | 12.4 | 21.1 | 17.15 | 17.1 |
| | 4 | 0 | 105.1 | 17.4 | 12.5 | 13.7 | 23.9 | 19.5 | 19.4 |
| | 4 | 6 | 110.0 | 18.3 | 13.2 | 14.45 | 25.8 | 20.8 | 20.65 |
| | 5 | 0 | 114.8 | 18.8 | 13.9 | 15.15 | 27.1 | 22.0 | 21.8 |
| | 5 | 6 | ..... | 19.9 | 14.5 | 15.65 | 28.2 | 22.9 | 22.7 |
| | 6 | 0 | 121.3 | 20.7 | 15.1 | 16.4 | 30.0 | 24.0 | 23.9 |
| | 6 | 6 | 125.5 | 21.1 | 15.5 | 16.9 | 30.8 | 24.9 | 24.7 |
| 29 | 3 | 3 | 101.7 | 16.2 | 12.0 | 13.2 | 21.4 | 17.4 | 17.2 |
| | 3 | 6 | 104.4 | 16.7 | 12.3 | 13.6 | 22.4 | 17.8 | 17.7 |
| | 4 | 0 | 107.0 | 17.4 | 12.8 | 14.2 | 23.3 | 18.8 | 18.7 |
| | 4 | 6 | 110.3 | 18.55 | 13.4 | 14.75 | 25.0 | 19.8 | 19.7 |
| | 5 | 0 | 113.3 | 19.2 | 13.9 | 15.3 | 26.0 | 20.7 | 20.5 |
| | 5 | 6 | 116.9 | 20.1 | 14.4 | 15.7 | 27.2 | 21.6 | 21.4 |
| 30 | 3 | 8 | ..... | 17.5 | 13.1 | 14.55 | 23.1 | 19.7 | 19.5 |
| | 4 | 3 | ..... | 18.3 | 13.8 | 15.3 | 24.3 | 20.8 | 20.6 |
| | 5 | 0 | 114.0 | 19.5 | 14.9 | 16.5 | 26.3 | 22.6 | 22.4 |
| | 6 | 6 | ..... | 22.2 | 16.5 | 18.4 | 30.0 | 25.7 | 25.3 |
| | 7 | 0 | 127.8 | 23.3 | 17.2 | 19.1 | 31.2 | 26.7 | 26.35 |
| | 7 | 6 | 130.9 | 23.1 | 17.6 | 19.6 | 32.1 | 27.6 | 27.2 |
| | 8 | 0 | 134.1 | 24.4 | 18.1 | 20.1 | 33.1 | 28.4 | 27.9 |
| 31 | 4 | 3 | 101.0 | 15.4 | 11.6 | 13.0 | 21.9 | 17.7 | 17.2 |
| | 5 | 0 | 106.8 | 16.4 | 12.4 | 13.8 | 23.5 | 19.2 | 18.6 |
| | 5 | 6 | 108.3 | 17.0 | 12.85 | 14.3 | 24.6 | 19.9 | 19.3 |
| | 6 | 0 | ..... | 17.6 | 13.4 | 14.9 | 25.6 | 20.9 | 20.2 |
| | 6 | 6 | 114.4 | 18.05 | 13.8 | 15.25 | 26.5 | 21.6 | 21.0 |
| | 7 | 0 | 117.9 | 18.8 | 14.3 | 15.7 | 27.4 | 22.3 | 21.7 |
| | 7 | 6 | 120.2 | 19.0 | 14.5 | 16.0 | 28.4 | 23.1 | 22.4 |

TABLE 5.—*Individual Data—Continued*

B. Girls—Continued

| Subject No. | Age | | Stature, Cm. | Lengths of Bones, Cm. | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Yr. | Mo. | | Humerus | Radius | Ulna | Femur | Tibia | Fibula |
| 32 | 4 | 3 | ..... | 17.0 | 12.9 | 13.9 | 23.5 | .... | .... |
| | 5 | 5 | 114.7 | 19.0 | 14.0 | 15.1 | 26.5 | 21.0 | 21.05 |
| | 6 | 3 | ..... | 20.1 | 14.7 | 16.0 | 28.4 | 22.4 | 22.4 |
| | 6 | 6 | 122.3 | 20.6 | 15.0 | 16.25 | 28.8 | 22.8 | 22.9 |
| | 7 | 0 | 125.0 | 21.3 | 15.4 | 16.7 | 29.6 | 23.5 | 23.5 |
| | 7 | 6 | 128.1 | 21.8 | 15.8 | 17.1 | 31.0 | 24.3 | 24.4 |
| | 8 | 0 | 131.5 | 22.6 | 16.3 | 17.6 | 32.2 | 25.2 | 25.3 |
| | 8 | 6 | 134.2 | 23.4 | 16.8 | 18.2 | 32.9 | 26.1 | 26.2 |
| | 9 | 0 | 136.7 | 24.1 | 17.1 | 18.6 | 34.2 | 27.1 | 27.0 |
| | 9 | 6 | 139.9 | 24.7 | 17.5 | 19.05 | 35.2 | 27.9 | 27.9 |
| | 10 | 0 | 142.8 | 25.2 | 18.0 | 19.5 | 36.6 | 28.6 | 28.6 |
| 33 | 4 | 6 | 100.5 | 16.2 | 12.3 | 13.6 | 22.9 | 18.7 | 18.5 |
| | 5 | 0 | ..... | 17.0 | 12.8 | 14.2 | 24.1 | 19.7 | 19.5 |
| | 5 | 6 | 108.0 | 17.7 | 13.2 | 14.7 | 25.2 | 20.5 | 20.3 |
| | 6 | 0 | 110.4 | 18.3 | 13.7 | 15.2 | 26.1 | 21.45 | 21.1 |
| 34 | 5 | 0 | 111.7 | 17.8 | 12.8 | 14.1 | 26.1 | 20.8 | 20.5 |
| | 5 | 9 | ..... | 18.9 | 13.8 | 14.9 | 27.8 | 22.2 | 22.0 |
| | 6 | 6 | 122.0 | 19.8 | 14.5 | 15.8 | 29.6 | 23.6 | 23.3 |
| | 7 | 0 | 125.7 | 20.0 | 15.0 | 16.4 | 30.4 | 24.5 | 24.2 |
| | 7 | 6 | 127.6 | 21.0 | 15.5 | 16.8 | 31.4 | 25.1 | 24.9 |
| | 8 | 0 | 129.8 | 21.7 | 16.1 | 17.4 | 32.4 | 26.1 | 25.9 |
| | 8 | 6 | 133.5 | 22.3 | 16.6 | 17.8 | 33.5 | 27.0 | 26.7 |
| | 9 | 0 | 137.0 | 23.2 | 17.2 | 18.4 | 35.0 | 27.7 | 27.6 |
| | 9 | 6 | 140.7 | 24.1 | 17.7 | 19.0 | 35.8 | 28.8 | 28.5 |
| 35 | 5 | 3 | ..... | 18.6 | 13.45 | 15.3 | 25.6 | 21.0 | 21.2 |
| | 5 | 10 | ..... | 19.6 | 14.0 | 16.0 | 26.5 | 22.0 | 22.2 |
| | 6 | 6 | 116.5 | 20.3 | 14.55 | 16.7 | 27.9 | 23.0 | 23.3 |
| | 7 | 0 | 120.1 | 20.4 | 15.1 | 17.1 | 28.8 | 23.8 | 24.0 |
| | 7 | 7 | 123.1 | 21.5 | 15.45 | 17.5 | 29.4 | 24.6 | 24.8 |
| | 8 | 0 | 125.0 | 22.1 | 15.8 | 18.0 | 30.3 | 25.3 | 25.6 |
| | 8 | 6 | 126.3 | 22.8 | 16.3 | 18.3 | 31.7 | 26.0 | 26.3 |
| | 9 | 0 | 128.9 | 23.0 | 16.5 | 18.6 | 32.2 | 26.7 | 26.9 |
| | 9 | 6 | 131.4 | 23.8 | 16.9 | 19.15 | 32.7 | 27.4 | 27.5 |
| 36 | 5 | 4 | ..... | 17.9 | 12.9 | 14.0 | 25.7 | 20.45 | 20.2 |
| | 5 | 7 | ..... | 18.4 | 13.1 | 14.3 | 26.3 | 20.9 | 20.5 |
| | 6 | 9 | 121.6 | 20.2 | 14.45 | 15.6 | 29.0 | 23.0 | 22.7 |
| | 7 | 6 | 127.8 | 21.3 | 15.2 | 16.55 | 30.3 | 24.3 | 23.9 |
| | 8 | 1 | 130.6 | 22.0 | 15.7 | 17.1 | 31.8 | 25.5 | 24.8 |
| | 8 | 6 | 132.6 | 22.5 | 16.0 | 17.45 | 32.3 | 26.2 | 25.6 |
| | 9 | 0 | 135.0 | 23.3 | 16.5 | 18.0 | 33.2 | 27.1 | 26.4 |
| | 9 | 6 | 138.5 | 23.6 | 16.9 | 18.4 | 34.6 | 28.0 | 27.0 |
| | 10 | 0 | 142.0 | 24.2 | 17.3 | 18.7 | 34.9 | 28.3 | 27.6 |
| 37 | 5 | 7 | 109.7 | 18.4 | 13.0 | 14.4 | 26.0 | 20.8 | 20.4 |
| | 6 | 6 | 117.1 | 19.8 | 14.0 | 15.4 | 28.4 | 22.6 | 22.0 |
| | 7 | 1 | 119.5 | 20.2 | 14.4 | 15.9 | 29.3 | 23.4 | 22.8 |
| | 8 | 0 | 124.6 | 21.5 | 15.3 | 16.9 | 31.1 | 24.9 | 24.4 |
| | 8 | 6 | ..... | 22.1 | 15.8 | 17.3 | 32.3 | 25.7 | 25.15 |
| | 9 | 0 | 131.0 | 22.9 | 16.3 | 17.85 | 33.5 | 26.6 | 26.05 |
| | 9 | 6 | 133.2 | 23.4 | 16.7 | 18.4 | 34.6 | 27.5 | 26.9 |
| | 10 | 0 | 137.0 | 24.1 | 17.1 | 18.9 | 35.4 | 28.6 | 27.8 |
| 38 | 5 | 8 | ..... | 18.1 | 13.5 | 14.9 | 26.4 | 21.0 | 21.0 |
| | 6 | 3 | ..... | 18.7 | 14.1 | 15.5 | 27.5 | 21.6 | 21.7 |
| | 7 | 0 | 118.2 | 19.3 | 14.7 | 16.2 | 29.2 | 23.0 | 22.9 |
| | 7 | 6 | 120.8 | 19.7 | 15.2 | 16.6 | 30.0 | 23.7 | 23.6 |
| | 8 | 0 | 123.8 | 20.6 | 15.55 | 17.1 | 31.1 | 24.5 | 24.4 |
| | 8 | 6 | 126.4 | 21.6 | 16.0 | 17.6 | 32.2 | 25.4 | 25.3 |
| | 9 | 0 | 129.3 | 22.4 | 16.5 | 18.2 | 33.1 | 26.3 | 26.1 |
| | 9 | 6 | 132.2 | 23.0 | 16.9 | 18.6 | 34.4 | 27.2 | 27.0 |
| | 10 | 0 | 135.4 | 23.3 | 17.35 | 19.1 | 34.9 | 28.0 | 27.7 |
| 39 | 5 | 9 | 107.2 | 18.3 | 12.9 | 14.7 | 25.6 | 21.2 | 20.2 |
| | 6 | 6 | 112.2 | 19.2 | 13.6 | 15.4 | 27.2 | 22.4 | 21.5 |
| | 7 | 3 | 116.7 | 19.8 | 14.2 | 16.1 | 28.7 | 23.7 | 22.8 |
| | 7 | 6 | 118.3 | 20.2 | 14.4 | 16.4 | 29.1 | 24.1 | 23.1 |
| | 8 | 0 | 120.3 | 20.9 | 14.8 | 16.9 | 30.2 | 25.0 | 23.9 |
| | 8 | 6 | 122.8 | 21.3 | 15.2 | 17.3 | 31.2 | 25.8 | 24.8 |
| | 9 | 0 | 126.9 | 22.2 | 15.7 | 17.8 | 32.1 | 26.8 | 25.6 |
| | 9 | 6 | 129.3 | 22.7 | 16.05 | 18.25 | 32.9 | 27.4 | 26.2 |
| 40 | 5 | 9 | ..... | 20.3 | 14.7 | 16.2 | 28.6 | 23.5 | 23.5 |
| | 6 | 10 | 127.7 | 21.5 | 15.8 | 17.6 | 30.5 | 25.5 | 25.4 |
| | 7 | 3 | 129.8 | 22.1 | 16.3 | 18.1 | 31.6 | 26.4 | 26.2 |
| | 8 | 0 | 134.7 | 23.1 | 17.0 | 18.8 | 33.4 | 27.9 | 27.6 |
| | 8 | 7 | 138.0 | 23.8 | 17.5 | 19.4 | 34.3 | 28.7 | 28.4 |
| | 9 | 0 | ..... | 24.1 | 17.9 | 19.9 | 35.2 | 29.6 | 29.3 |
| | 9 | 6 | 144.4 | 24.8 | 18.4 | 20.4 | 36.6 | 30.7 | 30.4 |
| | 10 | 0 | 147.7 | 25.3 | 19.0 | 20.9 | 37.3 | 31.9 | 31.4 |
| | 10 | 6 | 150.8 | 26.5 | 19.4 | 21.6 | 38.4 | 32.9 | 32.4 |

TABLE 5.—*Individual Data—Continued*

B. Girls—Continued

| Subject No. | Age | | Stature, Cm. | Lengths of Bones, Cm. | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | Yr. | Mo. | | Humerus | Radius | Ulna | Femur | Tibia | Fibula |
| 41 | 6 | 6 | ..... | 20.7 | 15.0 | 16.6 | 28.3 | 23.9 | 23.8 |
| | 7 | 9 | 129.8 | 22.4 | 16.4 | 18.05 | 31.4 | 26.3 | 26.2 |
| | 8 | 6 | 134.5 | 23.6 | 17.15 | 18.9 | 32.7 | 27.7 | 27.5 |
| | 9 | 0 | 137.5 | 24.5 | 17.7 | 19.5 | 33.8 | 28.8 | 28.5 |
| | 9 | 6 | 140.4 | 25.2 | 18.3 | 20.0 | 34.9 | 29.7 | 29.4 |
| | 10 | 0 | 144.4 | 25.8 | 18.8 | 20.6 | 36.2 | 30.9 | 30.4 |
| | 10 | 6 | 147.1 | 26.3 | 19.25 | 21.3 | 37.7 | 31.6 | 31.2 |
| | 11 | 0 | 150.0 | 27.4 | 19.8 | 21.75 | 38.2 | 32.6 | 32.1 |
| 42 | 6 | 8 | ..... | 19.5 | 14.5 | 16.0 | 29.4 | 23.2 | 23.2 |
| | 7 | 6 | 125.0 | 20.7 | 15.2 | 16.6 | 31.0 | 24.6 | 24.5 |
| | 8 | 0 | 128.5 | 21.3 | 15.8 | 17.2 | 32.1 | 25.5 | 25.4 |
| | 8 | 6 | 131.0 | 21.8 | 16.1 | 17.6 | 33.0 | 26.3 | 26.3 |
| | 9 | 0 | ..... | 22.3 | 16.7 | 18.2 | 34.3 | 27.3 | 27.3 |
| | 9 | 6 | 137.8 | 23.2 | 17.0 | 18.6 | 35.7 | 28.3 | 28.2 |
| | 10 | 0 | 142.5 | 23.9 | 17.6 | 19.3 | 36.6 | 29.5 | 29.2 |
| 43 | 6 | 9 | ..... | 20.5 | 15.3 | 17.0 | 28.5 | 24.1 | 24.1 |
| | 7 | 1 | 125.1 | 21.0 | 15.7 | 17.3 | 29.4 | 24.6 | 24.7 |
| | 7 | 6 | 128.5 | 21.6 | 16.1 | 17.8 | 30.4 | 25.5 | 25.4 |
| | 8 | 0 | 130.9 | 21.9 | 16.7 | 18.5 | 31.4 | 26.3 | 26.2 |
| | 8 | 6 | 135.4 | 22.9 | 17.1 | 18.9 | 32.3 | 27.2 | 27.1 |
| | 9 | 0 | 139.0 | 23.5 | 17.7 | 19.55 | 33.6 | 28.2 | 28.2 |
| | 9 | 6 | 141.7 | 24.4 | 18.05 | 19.9 | 34.5 | 29.0 | 28.9 |
| | 10 | 0 | 144.7 | 24.7 | 18.3 | 20.3 | 35.7 | 29.7 | 29.5 |
| | 10 | 6 | 147.2 | 25.4 | 18.8 | 20.9 | 36.0 | 30.5 | 30.2 |
| 44 | 6 | 9 | ..... | 21.0 | 15.8 | 17.3 | 29.5 | 25.0 | 24.6 |
| | 7 | 6 | 131.5 | 21.8 | 16.5 | 18.0 | 31.2 | 26.3 | 25.9 |
| | 8 | 0 | 134.2 | 22.6 | 17.0 | 18.5 | 32.4 | 27.2 | 26.7 |
| | 9 | 1 | 140.2 | 23.9 | 17.9 | 19.8 | 34.1 | 28.8 | 28.1 |
| | 9 | 6 | 142.4 | 24.7 | 18.4 | 20.0 | 34.7 | 29.9 | 29.2 |
| | 10 | 0 | 145.7 | 25.0 | 18.7 | 20.4 | 36.2 | 30.7 | 29.8 |
| | 10 | 6 | 148.4 | 25.6 | 19.1 | 20.9 | 36.6 | 31.3 | 30.5 |
| | 11 | 0 | 152.0 | 26.5 | 19.7 | 21.4 | 38.0 | 32.5 | 31.3 |
| 45 | 7 | 0 | 117.0 | 19.8 | 14.8 | 16.2 | 27.9 | 22.8 | 22.8 |
| | 7 | 5 | 119.7 | 20.4 | 15.1 | 16.7 | 28.9 | 23.5 | 23.3 |
| | 8 | 0 | 123.0 | 21.3 | 15.6 | 17.3 | 29.7 | 24.5 | 24.2 |
| | 8 | 6 | ..... | 21.8 | 16.1 | 17.7 | 30.8 | 25.3 | 25.0 |
| | 9 | 0 | 129.0 | 22.5 | 16.5 | 18.2 | 32.0 | 26.3 | 25.7 |
| | 9 | 7 | 131.2 | 23.0 | 17.0 | 18.9 | 33.0 | 26.9 | 26.4 |
| | 10 | 0 | 134.4 | 23.7 | 17.4 | 19.25 | 34.2 | 27.7 | 27.2 |
| 46 | 7 | 3 | 126.5 | 21.2 | 15.7 | 17.2 | 30.9 | 24.9 | 24.4 |
| | 8 | 0 | 131.2 | 22.3 | 16.5 | 18.0 | 32.5 | 26.2 | 25.6 |
| | 9 | 0 | ..... | 23.4 | 17.3 | 18.9 | 34.5 | 27.7 | 27.0 |
| | 10 | 1 | 141.5 | 24.7 | 18.0 | 19.9 | 35.9 | 29.2 | 28.6 |
| 47 | 7 | 7 | ..... | 23.1 | 16.9 | 18.6 | 34.0 | 27.5 | 27.2 |
| | 9 | 6 | 148.8 | 25.8 | 18.9 | 20.7 | 37.6 | 31.3 | 30.7 |
| | 10 | 7 | 157.9 | 26.6 | 20.3 | 22.5 | 41.6 | 33.8 | 32.7 |
| | 11 | 1 | 163.2 | 28.0 | 21.1 | 23.0 | 43.2 | 35.0 | 33.9 |
| | 11 | 6 | 166.9 | 29.5 | 21.9 | 24.5 | 44.15 | 35.8 | 35.0 |
| | 12 | 0 | 170.4 | 30.0 | 22.3 | 25.1 | 44.9 | 36.6 | 35.7 |
| 48 | 7 | 8 | ..... | 19.5 | 15.4 | 16.7 | 28.5 | 23.9 | 24.1 |
| | 9 | 0 | 123.6 | 21.2 | 16.5 | 17.9 | 30.7 | 26.1 | 26.1 |
| | 9 | 9 | 126.3 | 21.3 | 16.9 | 18.2 | 31.8 | 26.9 | 26.95 |
| | 10 | 0 | 127.4 | 21.7 | 17.1 | 18.5 | 32.3 | 27.2 | 27.3 |
| | 10 | 6 | 130.7 | 22.4 | 17.6 | 19.0 | 33.1 | 28.0 | 28.05 |
| | 11 | 0 | 131.9 | 22.8 | 18.0 | 19.4 | 34.0 | 28.9 | 28.8 |
| | 11 | 6 | 134.5 | 23.4 | 18.4 | 19.9 | 34.6 | 29.7 | 29.4 |
| | 12 | 0 | 138.0 | 23.9 | 18.85 | 20.3 | 35.4 | 30.3 | 30.1 |
| 49 | 8 | 2 | ..... | 20.9 | 15.3 | 17.2 | 31.8 | 25.9 | 25.8 |
| | 8 | 6 | 132.5 | 21.5 | 15.7 | 17.6 | 32.6 | 26.5 | 26.4 |
| 50 | 10 | 0 | 149.3 | 26.5 | 18.8 | 21.0 | 37.4 | 31.5 | 30.95 |
| 51 | 10 | 0 | 143.0 | 25.1 | 19.05 | 20.8 | 37.0 | 30.7 | 29.9 |
| | 10 | 6 | 146.4 | 25.8 | 19.6 | 21.4 | 37.5 | 31.6 | 30.7 |
| | 11 | 1 | 150.5 | 26.3 | 20.2 | 22.1 | 38.8 | 32.7 | 31.7 |
| | 11 | 6 | 154.4 | 27.2 | 20.7 | 22.5 | 40.0 | 33.6 | 32.6 |
| | 12 | 0 | 158.0 | 28.1 | 21.4 | 23.2 | 40.9 | 34.6 | 33.4 |
| 52 | 10 | 6 | 143.2 | 25.3 | 18.3 | 20.4 | 35.1 | 29.5 | 29.1 |
| | 11 | 0 | 146.3 | 25.9 | 18.9 | 20.6 | 35.7 | 30.3 | 29.9 |
| | 11 | 6 | 150.6 | 26.9 | 19.6 | 21.5 | 37.2 | 31.7 | 31.3 |
| | 12 | 0 | 154.5 | 27.6 | 20.0 | 22.5 | 38.3 | 32.3 | 31.9 |

Although nearly half the group of 90 children on whom sufficient data are available for study have some variation from the percentile zone trends, the general pattern for bone growth seems to be essentially uniform. The variations are small and become evident only when one is able to collect enough measure-

ments on a child over several years so that individual growth curves can be studied. I have no certain explanation as to why some of these growth rates are variable while others are so uniform. The children, although somewhat select from the socioeconomic and the intellectual standpoint, are probably quite representative of the upper middle class group. They are free from chronic disease and have had no more than the average number of acute illnesses. It is possible that unrecognized environmental factors may have influenced the growth rates of some. It is also certain that inherited characteristics of body build are playing a considerable part in determining the pattern of childhood growth. Some sex differences are definitely suggested in the present study, and the standards may well be too high for boys over at least part of this twelve year period.

An approximately straight line relationship between the length of one bone and that of another has been found, with high coefficients of correlation ranging from 0.9915 to 0.9990. Two of these correlations are shown in chart 7. High correlation coefficients are also found between stature and lengths of the bones. These range from 0.9913 to 0.9952. An essentially straight line relationship between these measurements exists after approximately 6 months of age, but prior to that time, as shown in chart 8, lengths of the bones are proportionately greater in relation to stature than they are at later ages. Thus, in the early postnatal months it is probable that the trunk is growing in length at a faster rate than the rest of the axial skeleton.

The changing relationships between length of the bones and stature with increasing age are well shown by the ratios calculated from these measurements, expressed as percentages in table 4 and illustrated in chart 9 for $\frac{\text{length of femur}}{\text{stature}}$ referred to age. The range of values at any age is seldom more than 2.5 per cent, and the range included in $\pm$ 1 $\sigma$ from the mean includes approximately 70 per cent of the values for each bone. On the other hand, ratios between the length of one bone and that of another in a given child are less indicative of changes in build; I feel that one is better able to detect variation in proportions between, for example, the length of the femur and the length of the humerus by comparing the lengths of these bones to percentile standards than by means of the $\frac{\text{femur}}{\text{humerus}}$ ratios.

In this preliminary report on the growth of the long bones of individual children I have not attempted to draw any certain or far reaching conclusions. I believe, however, that the method presented offers a reliable technic and one which deserves more extensive trial in the study of growth.

Colorado General Hospital.

# SYNECHIAS OF VULVA IN SMALL CHILDREN

H. E. BOWLES, M.D.
AND
L. S. CHILDS, M.D.
HONOLULU, TERRITORY OF HAWAII

The condition referred to in the following pages has been variously called synechias of the vulva, atresia of the vulva, vulvar fusion, occlusion of the vaginal vestibule, acquired gynatresia, adherent labia minora and other descriptive names. (The common adhesions about the clitoris analogous to preputial adhesions in males will not be discussed here.)

Considering that the anomaly is apparently common, the American literature is surprisingly mute on the subject. Most references occur in European articles. Griffith and Mitchell [1] remarked: "Atresia of the vulva is rare. There may be only epithelial adhesions of the greater or lesser labia, or there may be a firmer union, which if not complete will cause a retention of mucous secretions and later of menstrual discharges (hematocolpos), and if complete will also interfere with the passage of urine." Holt and McIntosh [2] stated: "Atresia of the vulva is not uncommon. It is usually due to delicate adhesions between the labia. As a rule the condition is not complete, a small opening permitting the urine to escape. These adhesions can be broken apart quite readily by separation of the labia; very little force is required, and an anesthetic is not necessary. If they are allowed to persist, dense adhesions may form." It is interesting to note that Campbell [3] in a summary of 9 cases of vulvar fusion observed by him remarked that the physicians who originally saw the patients thought they were dealing with hermaphrodites or pseudohermaphrodites. Familiarity with the entity will enable the attendant to escape ludicrous errors.

Failure to recognize the presence of labial synechias will occasionally result in a progressively thicker and tougher interlabial bridge, which may cause serious urinary and menstrual dysfunction or may preclude normal sex life. One of Campbell's patients, a child of 8 years, had been treated for recurrent pyelitis for nine months before a careful examination of the genitalia revealed a vulvar fusion.

## ETIOLOGY

There is much controversy as to whether synechias of the vulva are congenital or acquired. Taussig [4] stated that the agglutinations may result from various prenatal and postnatal conditions. Campbell expressed the opinion that in many cases the anomaly is congenital and not detected at the time of the child's birth. He stated that there is "an embryologic midline fusion of the labioscrotal folds on each side similar to that which the two scrotal folds undergo in the male, but the fusion usually lacks the density of scrotal fusion. This scrotal union occurs between the third and fourth months of fetal life."

1. Griffith, J. P. C., and Mitchell, A. B.: Textbook of Pediatrics, Philadelphia, W. B. Saunders Company, 1941, p. 705.
2. Holt, L. E., and McIntosh, B.: Holt's Diseases of Infancy and Childhood, ed. 11, New York, D. Appleton-Century Company, Inc., 1940, p. 813.
3. Campbell, M. F.: Vulvar Fusion, J. A. M. A. **115**:513 (Aug. 17) 1940.
4. Taussig, F. J., in Brennemann, J.: Practice of Pediatrics, Hagerstown, Md., W. F. Prior Company, Inc., 1942, vol. 3, p. 13.

Nowlin and Adams,[5] on the other hand, stated the belief that vulvar fusion is an acquired and not a congenital anomaly. They emphasized that in the development of the female genitalia each of the labia minora is developed separately from a genital fold. These genital folds are developed from the lips of a groove on the extremity of the genital tubercle, a structure which appears approximately in the fifth week of life. Since at no time of development is there fusion of the genital folds, one might assume that atresia of the vulva is not due to inhibition of normal development in the embryo as atresia of the vagina is.

Ochsenius[6] has stated that acquired vulvar fusion follows local irritation consequent to an acute infectious disease, such as cholera, smallpox, diphtheria, scarlet fever, pneumonia, typhoid or typhus. However, Campbell, in a thorough search has failed to find reports of a single authentic case of this variety. We have also been unable to do so.

In none of Nowlin and Adams' cases was there any history suggesting either a gonorrheal or a nonspecific vulvitis, and in none was vulvitis present at the time of examination. They remarked that it is probable that the fusion of the labia minora is favored by the richness in the cellularity of these parts in young girls plus the fact that these structures normally lie in close contact in early life, particularly in their posterior portion. As will be seen, our observations are in accord with those of Nowlin and Adams.

Vakar[7] expressed the belief that the desquamation normally occurring in the vulva and vagina predisposes to the penetration of the sensitive epithelium by pathogenic organisms. This may be; but bacteriologic study of the vagina and vulva is most unsatisfactory, and we are unable to concede that any single organism is to blame. There is usually no leukorrhea.

The opportunity of examining infants at birth and at subsequent intervals thereafter for routine health supervision enables these differences of opinion to be resolved. A tabular report of 20 cases is appended. Fifteen of the patients are known certainly to have acquired the condition after birth; 1 is definitely known to have had it at birth. In the other 4 it was noted by us at the time of the first visit. In none of the cases was there any associated severe illness, recognizable vulvitis or vaginitis or injury. The mothers of almost all the patients were reasonably intelligent and conscientious.

It is difficult to explain what causes these vulvar adhesions in well infants and children. About all that can be said is that whatever produces raw and cohesive surfaces, whether it is irritation from the friction of a diaper, ill fitting underwear, too zealous scrubbing by the overfastidious mother or masturbation, may predispose to the adhesions. It seems that bacteria or fungi play only a minor secondary role in the majority of cases. It seems also that the time-honored picket fence injury should be relegated to the background, although, interestingly, Taylor's[8] second patient, a 7 year old child, had a history of such trauma a year previous to the discovery of a firm bluish membrane nearly completely closing the vaginal orifice.

## INCIDENCE

The condition usually occurs in children a few months of age, although labial fusion has been noted in adults, as in Taylor's case.[8] His patient was a 74 year

5. Nowlin, P., and Adams, J. R.: Atresia of Vulva in Children, J. Pediat. **8**:200, 1936.
6. Ochsenius, K.: Interlabial Occlusion of Vulva in Children and Its Treatment, Deutsche med. Wchnschr. **53**:838 (May 13) 1927.
7. Vakar, N. R.: Vulvar Adhesions in Young Children, Pediatria **14**:477, 1930.
8. Taylor, W. N.: Vulvar Fusion: Two Cases with Urological Aspects, J. Urol. **45**:710, 1941.

old woman in whom the desquamation incidental to senile vulvovaginitis resulted in serious systemic disturbance precipitated by extensive synechias of the labia minora.

### SIGNS AND SYMPTOMS AND THEIR CLINICAL SIGNIFICANCE

In infants there are usually no symptoms. There is a deflection or interruption of the stream of urine, as shown in 6 of 8 of Nowlin's cases. With persistence of the unfreed adhesions, vesical tenesmus and infection of the urinary tract set in, as evidenced by fever, pain, pyuria and leukocytosis. Systemic damage may be severe, as in Taylor's case, in which incontinence and failing vision, severe pain in the lower part of the abdomen and inability to walk occurred in a 74 year old woman. In the diagnosis of true cystitis or pyelitis in the female it must be remembered that only a catheterized specimen of urine is trustworthy and that the anomaly will be found on catheterization.

### PROPHYLAXIS

The genitalia should be examined by the attending physician at birth or shortly after, and this procedure should be a part of each subsequent general physical examination. The mother should be instructed to follow this up by frequent inspection of the infant's genitalia. This cannot be done unless the child is lying quietly with the thighs widely separated, to permit deliberate inspection of the vulva. An assistant may be needed. The beginning adhesions can be easily separated at such a time. Gentle cleaning of the vulva is all important. Too much zeal will only create roughened surfaces which will adhere readily. Bland oil is usually recommended for cleansing. Whether this can cause maceration, and hence adhesions, is a matter for further study.

### ACTIVE TREATMENT OF THE ADHESIONS

As a rule treatment is extremely simple. In the majority of our cases firm avulsion of the labia with the child in the lithotomy position without anesthesia was sufficient. This avulsion can usually be accomplished easily if the operator wears dry rubber gloves and makes sure that the patient's skin is dry, so as to permit ready traction without slipping. Occasionally it is necessary to free the lowermost portion of the fused area by additional blunt dissection with a probe or hemostat. In cases in which union is thick and the patient older, some form of anesthesia may be needed, and sharp dissection with scissors or scalpel and suturing of raw edges are occasionally necessary. The raw surfaces must be continuously protected by a bland ointment, such as boric acid ointment or plain petrolatum, until epithelization is complete. Campbell has recommended leaving a small pledget of cotton between the labia to keep them separated for a short time. We have not found this necessary, and the procedure is open to some objection on the grounds that the pledget might be inadvertently poked up into the vagina by the child. Repeated application of the ointment and continued vigilance are necessary to forestall a fresh crop of adhesions.

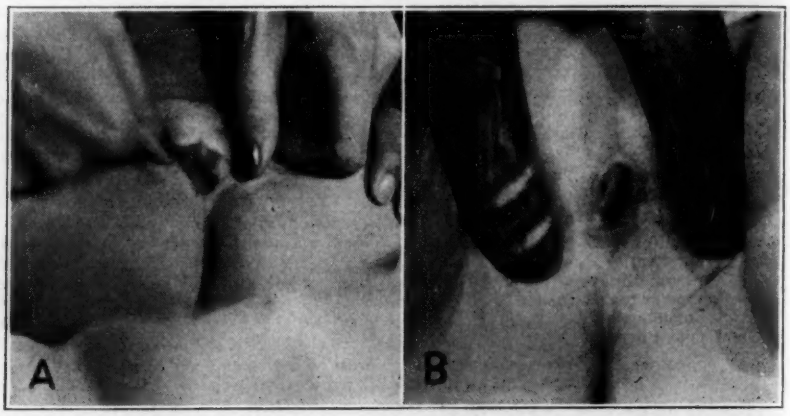
The 20 children whose condition is reported on in the appended table were all seen within a period of less than one year. Most of these children were delivered by the obstetric department of this clinic. Of the 20, only 1 was definitely known to have the anomaly at birth. Seven were delivered elsewhere, and the appearance of the infant's genitalia at the time of birth is not known. Fifteen acquired varying degrees of fusion between their periodic examinations in the outpatient pediatric service of the clinic.

| Subject | Age | Symptoms | Extent of Fusion of Labia Minora | Anesthetic | Treatment | Comment |
|---|---|---|---|---|---|---|
| L. K. | 7 mo. | None | Lower two thirds | None | Simple spreading of vulva | No fusion at birth or 36 days before this examination |
| M. M. | 3 mo. | None | Lower half | None | Simple spreading of vulva | No fusion at birth or 33 days before this examination |
| G. P. | 27 days | None | Lower half | None | Simple spreading of vulva | No fusion at birth or 10 days before this examination |
| D. B. | 4 mo. | None | Lower two thirds | None | Blunt dissection with tip of hemostat | Delivered elsewhere; said to have been normal at birth |
| M. B. | 23 mo. | None | Dense adhesions; almost complete fusion; small sinus at upper end | None | Spreading of labia and blunt dissection | Sister of D. B.; delivered elsewhere |
| A. K. | 3 yr. | Dysuria | Almost complete; small sinus at upper end | Nitrous oxide and oxygen | Separation of labia with tip of hemostat | Fusion noted at birth; uneventful healing |
| G. H. | 5 yr. | Bed-wetting | Almost complete | Light ether | Surgical incision | The complete protocol of this case follows |
| C. K. | 4 mo. | None | Lower half | None | Blunt dissection with tip of hemostat | No fusion at birth and 28 days before this examination |
| M. M. | 4 mo. | None | Lower half; very thin membrane | None | Simple spreading of vulva | No fusion at birth or 35 days before this examination |
| D. C. | 2½ yr. | None | Lower three fourths; found incidentally at bathing | None | Simple avulsion by spreading of adjacent tissues | Condition normal at birth and a few months before this examination |
| L. B. | 8 mo. | None | Practically complete, with 2 sinuses | None | Simple blunt dissection with tip of hemostat | The complete protocal of this case follows |
| M. L. B. | 5 mo. | Vomiting and night crying, 2 days; diffuse papular rash, possibly allergic, 1 week | Nearly complete, with tiny opening at upper end of fusion | None | Blunt dissection with hemostat | No fusion at birth or 44 days before this examination; spectacular subsidence of symptoms |
| P. S. | 6 mo. | None | Nearly complete, with pinhead size urinary sinus 0.5 cm. below clitoris | None | Stretching and blunt dissection | Delivered elsewhere |
| C. S. | 6 mo. | None | Nearly complete, from below upward | None | Simple spreading of labia | No fusion at birth or 44 days before this examination |
| N. A. | 17 mo. | None | Lowermost 1 cm. | None | Blunt dissection | No fusion at birth or 9 months before this examination |
| F. W. | 2 yr. | None | Nearly complete, from below upward; tiny urinary sinus just beneath clitoris | None | Blunt dissection with tip of hemostat | Delivered elsewhere; no fusion 36 days before this examination |
| C. A. | 5 mo. | None | Upper half | None | Blunt dissection with tip of hemostat | Delivered elsewhere; no fusion 27 days before this examination; this is the only instance in this series of anterior fusion of the labia minora |
| C. S. | 6 mo. | None | Almost complete; labia majora also adherent 2 cm. posteriorly | None | Blunt dissection | No fusion at birth or 47 days before this examination |
| F. H. | 10 mo. | None | Lower third; lightly adherent | None | Blunt dissection | No fusion at birth or 43 days before this examination |
| M. J. | 9½ mo. | None | Lower third; lightly fused | None | Blunt dissection | No fusion at birth or 83 days before this examination |

The cases of G. H. and L. B. merit further consideration.

The chief complaint of G. H., a girl aged 5 years, was bed-wetting. Although she had been under observation of physicians since the time of her birth, no data are available regarding the condition of her genitalia until she was 4½ years of age, when she was seen at a charity clinic. Her mother was told that the child had an imperforate hymen. On examination, the clitoris was found to be normal. About 0.5 cm. below this was a small circular orifice, through which the urine was voided. Labia could not be seen, and from this point down to where the fourchet would normally have been seen was thick, firm skin, giving the appearance of the complete absence of a vagina. With the child under light ether anesthesia an incision was made downward from the sinus beneath the clitoris toward the anus. A preliminary blunt dissection failed, as the tissue was too tough. Normal labia minora and a normal vagina were now revealed, and with the aid of a nasal speculum the cervix could be seen. A few interrupted fine plain catgut sutures were placed along the incised tissues on each side of the vulva and a petrolatum gauze dressing applied. Healing was rapid and complete with no residual deformity.

L. B., aged 8 months, showed no symptoms. She had been delivered elsewhere. Fusion of the labia was not present when she was last seen by us, sixty-five days before this examination. On examination there was revealed fusion of the labia minora with a small




Acquired partial occlusion of vulva: *A*, vulva of M. B.; *B*, vulva of G. H.

sinus 0.5 cm. below the clitoris. At this point another sinus, 2 mm. in diameter, was present, below which another adhesion extended to the fourchet. Only simple blunt dissection with the tip of a hemostat without anesthesia was required.

In all except 1 of our cases the fusion was firmest at the lowermost portion of the line of union. This would lead us to conclude that the union was oldest at this portion. Complete closure would probably occur if it were not for the stream of urine. The line of fusion was uninterrupted in all our subjects except L. B., in whom a gap of 1 cm. was present at the center of the line. In 1 subject (C. A.) the upper halves of the labia minora were fused and the urine dribbled through the lowermost portion of the vulva. In a few of these children new adhesions have tended to form possibly because of the mother's timidity. However, vigilance and frequent observation and the freeing of occasional new and lesser cohesions have resulted in successful cure in all cases.

One may well wonder what happens in the numerous cases of partial fusion of the labia minora which escape notice. We can only assume that most of the adhesions, being thin, are broken down by washing of the vulva, masturbation

or other mechanical process, much as is the case with preputial adhesions in the male. Salmond [9] and Hellier,[10] however, have reported cases in which the condition persisted into adult life.

## SUMMARY AND CONCLUSIONS

1. Fusion of the vulva of varying degrees, particularly in young girls, is a fairly common condition. Precise figures of incidence are lacking.

2. The condition may be rarely congenital. It is practically always acquired.

3. Vaginitis or acute illness was not noted in any case of our series.

4. In the absence of a specific etiologic agent, any minor trauma causing roughening of the surface of the labia is believed to be the most important factor.

5. The condition may occur in an adult, especially secondary to atrophic vulvovaginitis.

6. Symptoms and signs may be totally absent in adults as in the very young. In advanced stages serious pyelocystitis may occur, and if this is permitted to progress uremia may develop.

7. The adhesions usually occur from below upward. They may be interrupted by a short gap. As a rule, the lowermost portion of the fusion is more densely united than the upper. Often very short posterior synechias of only a few millimeters in length, are seen. We have not included cases of such adhesions in this series.

8. Treatment is usually simple and may be accomplished, as a rule, without anesthesia by simple spreading of the labia by the operator's hands with or without blunt dissection with the tip of a probe or hemostat. Incision with a sharp instrument under general anesthesia is occasionally indicated.

9. Faithful follow-up care with the use of a bland ointment is usually all that is necessary. Continued vigilance is needed to forestall fresh adhesions.

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9. Salmond, M.: Case of Atresia Vulvae Superficialis, J. Obst. & Gynaec. Brit. Emp. **37**:824, 1930.

10. Hellier, J. B., cited by Salmond.[9]

# BLOOD DIASTASE IN MUMPS

JAMES P. MURPHY, M.D.

GEORGE S. BOZALIS, M.D.

AND

EARL J. BIERI, M.D.

ST. LOUIS

Somogyi [1] and Gray, Probstein and Heifetz [2] recently reviewed the subject of blood diastase and its clinical significance. The latter authors made the statement [2b]: "Elevated diastase values have been found in acute infection of the parotid gland, including some cases of mumps.[3] In a small series of cases of mumps at the St. Louis City Isolation Hospital, no elevation of the blood diastase was found." We felt, however, that the extreme swelling of the parotid glands and their ducts, closing off the secretion from the mouth, would probably lead to rupture of the parenchymatous cells. The escape of the enzyme into the interstitial tissue and its subsequent absorption into the blood stream should lead to an increase in blood diastase at some time during the disease. Somogyi [1] has expressed the same idea, and Polacco and Midana [4] have shown it experimentally.

Accordingly, 35 patients with mumps, of all ages, of both sexes, with and without complications and at various periods of the disease, were studied. For many patients more than one determination was obtained.

The method used was Somogyi's [5] determination of diastase activity. None of the patients had any clinical evidence of transient acute pancreatitis,[2b] perforating posterior peptic ulcer [6] or retention of urine.[2b] In a control series of 60 determinations on 20 subjects with primary and with secondary syphilis, no reading was obtained above 180; many were in the 30 to 50 range.

Our results are listed in the table.

The normal range of blood diastase activity according to Somogyi [1] is 80 to 150, with 150 to 180 as a sort of zone of suspicion, or zone of high normal values. In the 58 determinations on the 35 patients only 7 values were below 200. Four of these were for patients who had other readings that were well above 200. In only 3 instances were the figures below 200: patient 12, 127; patient 16, 175 and patient 22, 140. Unfortunately, for these 3 patients only 1 determination of diastase was performed. To judge by the fluctuations of the diastase levels in

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From the St. Louis City Isolation Hospital.

1. Somogyi, M.: Diastatic Activity in Human Blood, Arch. Int. Med. **67**:665-679 (March) 1941.

2. (*a*) Gray, S. H.; Probstein, J. G., and Heifetz, C. J.: Clinical Studies on Blood Diastase, Arch. Int. Med. **67**:805-818 (April) 1941. (*b*) Heifetz, C. J.; Probstein, J. G., and Gray, S. H.: Clinical Studies on Blood Diastase, ibid. **67**:819-827 (April) 1941.

3. Dunlop, G. A.: The Diastatic Index in Acute Parotitis, Lancet **2**:183, 1933. Branistéanu, D., and Boutroux, A.: Contribution à l'étude de l'élimination de la amylase urinaire dans divers cas normaux et pathologiques, Arch. d. mal. de l'app. digestif. **23**:746, 1933.

4. Polacco, E., and Midana, A.: Influenza della ligatura die dotti parotidei sulla amilasi del sangue e dell'orina in cani spancreati e non, Arch. ed atti d. Soc. ital. di chir. **35**:654, 1929.

5. Somogyi, M.: Micromethods for the Estimation of Diastase, J. Biol. Chem. **125**:399, 1938.

6. Probstein, J. G.; Wheeler, P. A., and Gray, S. H.: Perforated Peptic Ulcer: Its Differentiation from Acute Pancreatitis by Blood Diastase Determinations, J. Lab. & Clin. Med. **24**:449, 1939.

all our patients and the higher values obtained in other determinations on the blood of the 4 other patients for whom we obtained low readings, we feel justified in assuming that had more tests been done on these 3 patients higher diastase

*Diastase Activity of the Blood of Patients with Mumps*

| Subject | Age, Yr. | Glands Involved | Estimated Diastase Activity | Day of Disease |
|---|---|---|---|---|
| 1 | 18 | Right parotid | 280 | 8 |
| | | | 350 | 10 |
| | | | 254 | 12 |
| | | | 175 | 16 |
| 2 | 20 | Bilateral parotid | 933 | 8 |
| 3 | 6 | Bilateral parotid | 4,000 | 8 |
| | | | 1,750 | 11 |
| 4 | 24 | Bilateral parotid | 933 | 5 |
| 5 | 29 | Right parotid | 200 | 3 |
| 6 | 11 | Right parotid | 215 | 7 |
| 7 | 5 | Left parotid | 1,400 | 3 |
| | | | 933 | 5 |
| 8 | 47 | Bilateral parotid | 1,000 | 4 |
| 9 | 18 | Bilateral parotid | 535 | 3 |
| | | | 519 | 7 |
| | | | 400 | 9 |
| 10 | 18 | Bilateral parotid | 410 | 3 |
| | | | 360 | 7 |
| | | | 163 | 9 |
| 11 | 32 | Bilateral parotid | 305 | 6 |
| | | | 465 | 9 |
| 12 | 39 | Left parotid | 127 | 6 |
| 13 | 6 | Left parotid | 240 | 4 |
| 14 | 18 | Bilateral parotid | 466 | 4 |
| | | | 280 | 7 |
| 15 | 17 | Left parotid | 360 | 4 |
| | | | 540 | 7 |
| 16 | 19 | Right parotid | 175 | 4 |
| 17 | 17 | Bilateral parotid | 317 | 3 |
| | | | 440 | 9 |
| 18 | 18 | Left parotid, right submaxillary | 583 | 5 |
| 19 | 22 | Right submaxillary | 700 | 3 |
| 20 | 24 | Bilateral parotid | 411 | 5 |
| 21 | 18 | Right submaxillary | 516 | 4 |
| | | | 700 | 8 |
| 22 | 19 | Left parotid | 140 | 5 |
| 23 | 21 | Right parotid | 775 | 5 |
| | | Right submaxillary | 250 | 13 |
| 24 | 18 | Bilateral parotid | 935 | 4 |
| 25 | 24 | Right parotid | 140 | 1 |
| | | | 467 | 4 |
| 26 | 21 | Left parotid | 875 | 5 |
| | | | 800 | 8 |
| | | | 379 | 11 |
| 27 | 17 | Right parotid | 500 | 6 |
| | | Right submaxillary | 240 | 13 |
| | | | 127 | 15 |
| 28 | 21 | Left parotid | 500 | 4 |
| | | | 539 | 10 |
| 29 | 18 | Right parotid | 451 | 8 |
| | | | 341 | 12 |
| 30 | 19 | Left parotid | 875 | 8 |
| | | | 225 | 13 |
| 31 | 21 | Bilateral parotid | 200 | 7 |
| 32 | 26 | Right parotid | 1,066 | 8 |
| 33 | 22 | Bilateral parotid | 933 | 7 |
| 34 | 27 | Left submaxillary | 233 | 11 |
| 35 | 18 | Left parotid | 318 | 9 |

levels would have been found. It can be stated, however, that in the overwhelming majority of cases of mumps the blood diastase is elevated.

Since there were only 5 females among the 35 patients (patients 5, 6, 20, 25 and 34), we cannot attach any significance to the fact that none of them had an

unusually high diastase reading (above 500). Of these 5, 4 had only single determinations.

Complications (in patients 1, 2, 4, 9, 11, 12 and 13) such as orchitis seemed to play no role in the diastase levels.

Cervical adenopathy, which may simulate mumps clinically, would fall in the group of localized infections that tend to give low diastase values.[2a] Thus, there is a simple laboratory procedure that would aid materially in differentiating between these diseases.

## CONCLUSION

The blood diastase activity is increased in mumps.

1515 Lafayette Avenue.

# Case Reports

## ACUTE INFECTIOUS LYMPHOCYTOSIS

PETER A. DUNCAN, M.D.
NEW YORK

In 1941 Smith[1] reported two types of lymphocytic reaction of the blood occurring in childhood which he designated as acute and chronic infectious lymphocytosis. These could be differentiated hematologically from infectious mononucleosis and leukemia, with which they were most often confused. A relative and absolute increase in the number of lymphocytes was common to both forms of the disease, and well defined mature lymphocytes with deeply basophilic cytoplasm characterized the blood smear.

The acute form of the disease occurred infrequently and was associated with a striking leukocytosis of short duration without recognizable symptoms and with a minimum of physical signs. The chronic type was more frequently encountered and followed infections of the upper respiratory tract of varying severity and was accompanied by low grade fever which persisted for weeks or months. Associated symptoms frequently included anorexia, pallor, fatigability and abdominal pain which was usually referred to the region of the umbilicus.

In the following report is described a case of acute infectious lymphocytosis which in addition to the striking blood picture also presented unusual signs and symptoms at the onset.

### REPORT OF A CASE

E. N., a white girl aged 4 years 11 months, was seen in the emergency room with a chief complaint of abdominal pain on Aug. 17, 1942.

*History.*—The family history was noncontributory. The birth, nutritional history and development of the patient were entirely normal. At the age of 1 year she had had pertussis. She had had chickenpox when 3 years old. Except for an occasional cold, her health had been excellent. She had received single monthly injections "against diphtheria" the two months preceding her admission to the hospital.

The patient had been in her usual good health until the evening before admission. She had attended the movies in the afternoon with her parents. Afterward, she was carried home because she was "tired." She went directly to bed and fell asleep immediately. During the night she fell out of bed to the floor without apparent injury. She had no complaints at this time and soon was asleep again. Awakening at her usual time, the child complained of abdominal pain. This pain persisted steadily for several hours and seemed to be increasing in severity. During this interval she "retched" on one occasion but did not vomit.

The patient was seen in the hospital at 10 a. m. She was extremely irritable and appeared acutely ill. The temperature was 99.6 F. The chief abnormal condition noted on examination was a boardlike rigidity and tenderness of the entire abdomen. The possibility of an acute condition of the abdomen requiring surgical intervention was considered, and a blood count was obtained. When this revealed a total white cell count of 45,600 with 65 per cent lymphocytes, the child was admitted to the pediatric service with a diagnosis of acute lymphatic leukemia.

*Physical Examination.*—The patient was a well developed and well nourished child appearing listless. She preferred to lie on her side with her neck and thighs flexed. The skin was hot and dry. The temperature on admission was 101.4 F. Respirations were rapid and shallow. The lips and mucous membranes were dry. The pharynx was mildly inflamed. The tonsils were moderately enlarged and cryptic. Very small cervical and inguinal lymph nodes were palpable. In the left axilla, a moderately enlarged, firm, nontender node was felt. There was a pronounced generalized rigidity of the abdominal musculature. She complained

From the Pediatric Service, St. Luke's Hospital, service of Dr. Herbert F. Jackson.

1. Smith, C. H.: Infectious Lymphocytosis, Am. J. Dis. Child. **62**:231 (Aug.) 1941.

of generalized abdominal tenderness to direct palpation. Rebound tenderness was not present. The remainder of the examination revealed essentially normal conditions.

*Course.*—The patient slept most of the afternoon of the day of admission. At 4 p. m. the temperature was 101.6 F. She complained of generalized abdominal pain and a frontal headache. After a fairly good supper, she became cheerful and alert. The abdominal pain had entirely disappeared, but a mild headache persisted. Examination of the abdomen at this time revealed that it was completely relaxed and soft. The liver and spleen could not be palpated. No other masses were present. During her sleep that night she occasionally cried out. The temperature had fallen gradually to normal during the night and remained normal the entire next day. During her second day in the hospital she still complained of headache. She vomited nine times, several of which were projectile. She slept a good deal during the day. At night, she awoke on several occasions for short periods. The vomiting stopped suddenly on her third day in the hospital. She complained of abdominal pain for a short period in the late afternoon. During the night, the temperature gradually rose; it reached a peak of 103 F. at 4 p. m. of her fourth day in the hospital. On awakening that morning, she had been irritable and had complained of generalized abdominal pain. Nausea was present, but vomiting did not occur. There was voluntary rigidity of the abdominal muscles, but palpation was possible and nothing unusual was noted. Tenderness was not present. The abdominal pain disappeared in the afternoon, but the child was listless and slept a great deal. The temperature gradually decreased, reaching normal the following day.

The patient remained in the hospital for forty-nine days. Throughout the rest of her stay, she was asymptomatic. She became active and ate well but still appeared somewhat pale. The temperature on most occasions ranged between 99 and 100 F. The lymph glands remained as at the time of admission. The liver and spleen was never palpated.

She was discharged to a convalescent home, where she stayed for one month. She had one short bout of abdominal pain while she was there. The temperature was constantly between 99 and 100 F.

On November 4, she was readmitted to the hospital for further study. The physical examination still showed the lymph nodes to be as noted on her previous admission, but this was the only abnormality observed. The course of her temperature was not remarkable, and the patient remained asymptomatic. She was discharged on Nov. 12.

Since her discharge, the patient has been followed in the outpatient department. She has remained asymptomatic, and the results of physical examinations have been essentially normal.

*Laboratory Data.*—The hematologic data will be discussed in a separate section. On August 28, a sternal marrow puncture was done. The differential cell count of a smear of the bone marrow showed: nucleated red blood cells per hundred white blood cells, 3; lymphocytes, 86 per cent; polymorphonuclear leukocytes, 5 per cent; myelocytes, 4 per cent; myeloblasts, 3 per cent, and eosinophils 2 per cent.

Microscopic study of sections of the bone marrow showed scattered red blood cells, lymphoid cells and neutrophilic and eosinophilic polymorphonuclear cells. The predominant type of cell was the small lymphocyte. Three roentgenograms of the chest showed no abnormalities. Repeated examinations of the urine gave results within normal limits. The Schick, Mantoux (1:10,000) and Kline reactions were negative. The bleeding and coagulation times and clot retraction were normal on two occasions. A heterophile agglutination (Paul-Bunnell) test gave a negative reaction on August 19.

*Hematologic Data.*—A complete blood count, including a platelet count, was done every other day during her first stay in the hospital and at varying intervals in the follow-up study. The results of the determinations during the first stay in the hospital are presented in the table. The changes noted are based on the normal values for the blood of patients of this child's age group, as discussed by Smith.[1]

A. Total Leukocyte Count: The initial leukocyte count, of 45,600, continued to rise rapidly to reach 66,500 in the late afternoon of the day of admission to the hospital. A peak of 110,000 was obtained on the third day in the hospital. Two days later, the total leukocyte count had fallen sharply to 38,100. For the next seventeen days, the number of white blood cells ranged chiefly from 30,000 to 45,000. There was then a gradual decrease in the total count until the higher limit of normal was reached on the thirty-fifth day in the hospital. In a follow-up study from that time, the total white cells have been consistently within normal limits.

B. Percentage and Total Number of Lymphocytes: The percentages of lymphocytes in the two determinations on the first day in the hospital were 65 and 68 respectively. By the third day, lymphocytes comprised 92 per cent of the white blood cells, as shown by a differential count. Percentages of 95 and 93 were recorded on the fifth and seventh days respectively. High levels

in the percentage of lymphocytes were maintained for the next twenty days, usually between 80 and 90 per cent. On the thirty-first day in the hospital, a rapid decrease in the percentage of lymphocytes was noted, falling to the higher limits of normal. For the following three weeks, the percentage of lymphocytes was usually between 45 and 50. During the follow-up study the lymphocytes ranged from 24 to 48 per cent.

The total number of lymphocytes during the patient's course fluctuated in a manner similar to that previously described for the total leukocyte count.

C. Percentage and Total Number of Polymorphonuclear Neutrophils: The neutrophils in the differential smear dropped rapidly from the 30 per cent determined on admission to 3 per cent on the fifth day in the hospital. The percentage of these cells remained at low levels, usually between 12 and 18, until a gradual rise to a normal level was noted on the twenty-fifth day.

An elevated count of 13,680 polymorphonuclear neutrophils was noted on the child's admission to the hospital. This increased to 18,620 on the afternoon of the same day. A sharp decrease in the total number of neutrophils then occurred, the count reaching 1,143 on the fifth day

*Blood Counts*

| Date | White Cells, Number per Cu. Mm. | Lymphocytes | | Neutrophils | | Platelets, Number |
|---|---|---|---|---|---|---|
| | | Percentage | Number | Percentage | Number | |
| 8/17 (a. m.) | 45,600 | 65 | 29,640 | 30 | 13,680 | |
| 8/17 (p. m.) | 66,600 | 68 | 45,220 | 28 | 18,620 | 140,000 |
| 8/19 | 110,000 | 92 | 101,200 | 7 | 7,700 | 120,000 |
| 8/21 | 38,100 | 95 | 36,195 | 3 | 1,143 | 110,000 |
| 8/23 | 38,700 | 93 | 35,991 | 5 | 1,935 | 100,000 |
| 8/25 | 36,800 | 81 | 29,808 | 11 | 4,048 | 90,000 |
| 8/27 | 46,900 | 86 | 40,334 | 10 | 4,690 | 400,000 |
| 8/29 | 30,000 | 72 | 21,600 | 25 | 7,500 | 370,000 |
| 8/31 | 42,600 | 89 | 37,914 | 9 | 3,834 | 400,000 |
| 9/ 2 | 28,300 | 84 | 23,772 | 13 | 3,679 | 410,000 |
| 9/ 4 | 34,700 | 85 | 29,495 | 12 | 4,164 | 350,000 |
| 9/ 6 | 36,600 | 85 | 31,110 | 12 | 4,392 | 430,000 |
| 9/ 8 | 27,200 | 80 | 21,769 | 18 | 4,896 | 110,000 |
| 9/10 | 28,100 | 86 | 24,166 | 13 | 3,653 | 310,000 |
| 9/12 | 19,900 | 79 | 15,721 | 18 | 3,582 | 410,000 |
| 9/14 | 22,000 | 66 | 14,520 | 25 | 5,500 | 140,000 |
| 9/16 | 18,100 | 47 | 8,507 | 39 | 7,059 | 420,000 |
| 9/18 | 23,700 | 45 | 10,665 | 41 | 9,717 | 210,000 |
| 9/20 | 13,500 | 55 | 7,425 | 39 | 5,265 | 380,000 |
| 9/22 | 12,400 | 56 | 6,944 | 36 | 4,464 | 280,000 |
| 9/24 | 8,100 | 50 | 4,050 | 48 | 3,888 | 130,000 |
| 9/26 | 12,700 | 44 | 5,588 | 49 | 6,223 | 220,000 |
| 9/28 | 10,100 | 49 | 4,949 | 38 | 3,838 | 415,000 |
| 9/30 | 11,000 | 39 | 4,290 | 46 | 5,060 | 320,000 |
| 10/ 2 | 7,900 | 53 | 4,187 | 28 | 2,212 | 310,000 |
| 10/ 4 | 7,800 | 46 | 3,588 | 40 | 3,120 | 310,000 |

in the hospital. There were 1,935 neutrophils on the seventh hospital day. By the ninth day, this count had risen to 4,048. Thereafter, the total polymorphonuclear neutrophil count usually fluctuated around the normal range, of 4,000 to 5,100, with one exception. There was a transitory increase beginning on the twenty-seventh day in the hospital, reaching 9,717 on the thirty-third day and returning to normal by the thirty-fifth day. This rise corresponded to the period of sharp decline in the percentage of lymphocytes in the differential count.

D. Erythrocytes and Hemoglobin Content: With the exception of a mild normocytic anemia, nothing unusual was noted with respect to the number of red blood cells or the hemoglobin content throughout the course of the patient's illness. There was a slight variation of the erythrocytes about an average of 4,000,000. The hemoglobin content showed a slight fluctuation about 11 Gm. per hundred cubic centimeters.

E. Platelets: The number of platelets dropped gradually from 140,000 on admission to 90,000 on the ninth day in the hospital. From the eleventh to the twenty-first day, the count varied from 350,000 to 430,000. During the remainder of the illness and throughout the follow-up period, there was a wider fluctuation, but in the majority of determinations the number was above 250,000.

F. Morphology: The structure of the red blood cells was normal except for a slight hypochromia. Platelets were always present in the smear. The myeloid cells, except for their scarcity, were normal. The predominant type of cell was the mature small lymphocyte. No immature or atypical lymphocytes were seen at any time.

## COMMENT

As already stated, Smith,[1] in 1941, described 2 cases of a condition which he designated as acute infectious lymphocytosis. He expressed the belief that the group of cases reported by Reyersbach and Lenert [2] and 1 case reported by Wilson and Cunningham [3] belonged in this classification rather than among the cases of infectious mononucleosis, as reported.

In all these cases there were no significant clinical signs or symptoms. The condition was detected fortuitously by a blood count taken for some other reason or as a routine institutional procedure. It was classified as a new syndrome on the basis of the hematologic observations. The chief diagnostic feature was a relative and absolute increase in the number of small mature lymphocytes. The duration of the lymphocytosis averaged four to five weeks. The disease was differentiated from infectious mononucleosis by the higher total white blood cell count, the absence of atypical lymphocytes, the negative result of a heterophil agglutination test and the absence of clinical signs and symptoms usually associated with that disease. The absence of immature white blood cells and a progressive anemia and thrombocytopenia served to distinguish infectious lymphocytosis from lymphatic leukemia. Examination of the bone marrow in Smith's cases showed an increased number of small lymphocytes in the differential count but no increase in the immature forms, so uniformly present in leukemia. The absence of immature forms in the blood smear differentiated infectious lymphocytosis from leukemoid reactions. The lymphocytosis of specific infectious diseases was ruled out by the lack of a history and of clinical evidence of these diseases.

The hematologic data in the present case were similar to those described by Smith.[1] There was a relative and absolute lymphocytosis of approximately five weeks' duration. Small mature lymphocytes were responsible for this increase. Neither atypical nor immature lymphocytes were observed throughout the course of the disease. Additional hematologic changes were observed early in the course of the present case. An initial myeloid stimulation was indicated by an increase of the total neutrophil count on the first day. However, this dropped rapidly to extremely low levels on the fifth and seventh days; this may indicate some suppression of the myeloid tissue at those times. The persistently low platelet count during the first nine days added further evidence of myeloid suppression during this period.

In contrast to the absence of clinical signs and symptoms in the previously reported cases, in the present case there was a dramatic onset. The abdominal pain, tenderness and rigidity first suggested an acute condition within the abdomen indicating surgical intervention. It was not until the results of the blood count were obtained and verified that a dyscrasia of the blood was suspected. These acute abdominal symptoms were present for at least ten hours and then rapidly subsided. The abdominal pain returned in severe form for a short interval on the

2. Reyersbach, G., and Lenert, T. F.: Infectious Mononucleosis Without Clinical Signs or Symptoms, Am. J. Dis. Child. **61**:237 (Feb.) 1941.

3. Wilson, C. P., and Cunningham, R. S.: Consideration of Supra-Vital Method of Studying Blood in Cases of Mononuclear Cell Response, Folia haemat. **38**:14 (March) 1929.

fourth day in the hospital. The abdominal pain may possibly be explained on the basis of hyperplasia of the mesenteric lymph nodes.[4]

Recently, Thelander and Shaw [5] have reported 2 cases of a condition described as meningitis associated with the blood picture of infectious mononucleosis. The patients presented signs of meningeal irritation at the onset of their disease. Examination of their spinal fluids revealed 40 and 90 cells per cubic millimeter, of which lymphocytes comprised 37 and 26 per cent respectively as the only abnormal findings. The blood picture in these cases showed white cell counts of 50,500 and 45,700. Lymphocytes comprised 81 per cent of the cells in the differential smear at the height of the leukocytosis. The structure of the lymphocytes in the blood smear was reported as typical of infectious mononucleosis. Other clinical signs and symptoms of infectious mononucleosis were absent, and the heterophil agglutination test gave a negative reaction. It is my opinion that these patients had acute infectious lymphocytosis with involvement of the central nervous system rather than infectious mononucleosis, as reported. In retrospect, the symptoms of irritability, headache, lethargy, nausea and projectile vomiting noted during the first week of my patient's illness would seem to indicate involvement of the nervous system. Unfortunately, an examination of the spinal fluid was not carried out.

In previously reported cases of acute infectious lymphocytosis, the patients have been afebrile throughout the course of the illness. An irregular febrile reaction occurred during the first week in the present case. It reached a peak of 103 F. on the fourth day. Throughout the rest of the course of the disease the patient was essentially afebrile.

The cause of infectious lymphocytosis remains obscure. The cases of the condition observed by Reyersbach and Lenert [2] in patients in a convalescent home indicated its infectious nature. Their data suggested a long incubation period and low communicability. Their investigations showed that Listerella monocytogenes, known to cause a relative and absolute lymphocytosis when injected into rabbits,[6] was not involved in their cases. No special bacterial investigations were carried out in the present case. Routine cultures of materials from the nose and throat contained a normal flora. It is my belief that etiologically this infection falls into the ever increasing group of virus infections which at present are not readily detectable by the routine laboratory procedures.

## SUMMARY

A case of acute infectious lymphocytosis is presented. The hematologic findings were similar to those described by Smith.[1] In previously reported cases the disease has been without signs or symptoms. In the case described here pronounced abdominal signs and symptoms were present at its onset, and evidences of possible involvement of the nervous system were noted during the first week of the disease. Two cases from the literature are reviewed as cases of acute infectious lymphocytosis with proved changes in the spinal fluid.

St. Luke's Hospital.

4. Sobel, I. P., and Stetten, DeW.: Nonspecific Mesenteric Lymphadenitis: Report of Fifty-Eight Cases, J. Pediat. **17**:305 (Sept.) 1940.

5. Thelander, H. E., and Shaw, E. B.: Infectious Mononucleosis with Special Reference to Cerebral Complications, Am. J. Dis. Child. **61**:1131 (June) 1941.

6. Murray, E. G. D.; Webb, R. A., and Swann, M. B. R.: A Disease of Rabbits Characterized by a Large Mononuclear Leucocytosis, Caused by a Hitherto Undescribed Bacillus, Bacterium Monocytogenes (n. sp.), J. Path. & Bact. **29**:407 (Oct.) 1926.

# CONGENITAL ATRESIA OF THE DUODENUM AND OF THE ILEUM

## REPORT OF TWO CASES WITH SUCCESSFUL RESULTS FOLLOWING OPERATION

EDWIN M. MILLER, M.D.

JOSEPH GREENGARD, M.D.

AND

WILLIAM B. RAYCRAFT, M.D.

CHICAGO

AND

IRMA McFADDEN, M.D.

DUBUQUE, IOWA

It is not necessary, it is even a grave mistake, for a physician or for a parent to feel that a newborn infant presenting evidence of a complete obstruction of the small bowel offers a hopeless problem. While the prognosis cannot be spoken of as "good," with early diagnosis and prompt surgical management there is a fair chance of a complete recovery. Indeed, this field of endeavor offers a thrilling challenge to the diagnostic ability of the pediatrician and the technical ability of the surgeon.

Congenital lesions of the gastrointestinal tract producing obstruction at birth are most apt to occur at one of four sites: (1) the esophagus at about the level of the bifurcation of the trachea; (2) the duodenum close to the ampulla of Vater; (3) the lower portion of the ileum in the region of Meckel's diverticulum, and (4) the lower part of the rectum and the anal orifice. In other words, they are most likely to be found (as Bland Sutton said more than fifty years ago) at those places where the embryologic processes are the most complex and therefore the chances of error in development the greatest. It is our purpose here to confine our discussion to the congenital obstructions of the duodenum and the small bowel, with the particular object of contrasting the two clinical pictures, of emphasizing the essential points in the treatment of both and finally of adding to the extremely meager list of successful results 2 cases of congenital atresia of the ileum in which we have performed successful operations within the past two years.

### CONGENITAL OBSTRUCTION OF THE DUODENUM

The problem of congenital obstruction of the duodenum is rapidly becoming more interesting and more encouraging, for successful results following surgical intervention are more frequently being reported. The pathology is well understood, because up to within the last twenty-five years the abundant literature on this subject was an uninterrupted series of autopsy reports, and not until 1916 was the first report of operative success published by Ernst,[1] of Copenhagen. In general, two types of lesions are recognized: the intrinsic, consisting either of a narrowing of the lumen (stenosis), usually at or near the ampulla of Vater, or of a complete interruption of the continuity of the bowel (atresia); occasionally an iris-like diaphragm or partition (such as that depicted in the cases reported by

1. Ernst, N. P.: A Case of Congenital Atresia of the Duodenum Treated Successfully by Operation, Brit. M. J. **1**:644, 1916.

Seidlin [2] and Morton and Jones [3]) is found which effectively causes a blocking of the channel. The extrinsic variety is due to something outside the bowel wall, such as a congenital band (Jackson [4]), pressure from overlying vessels (superior mesenteric), and very commonly, as pointed out in the writings of Prof. William E. Ladd,[5] of Boston, and Dr. E. J. Donovan, of New York,[6] a twist, or volvulus, of the midgut on the axis of the superior mesenteric artery, associated with nonrotation, incomplete rotation or reversed rotation of the large bowel. Whatever type of pathologic change may be present to cause the obstruction, the clinical picture is quite the same, and if the baby is carefully observed the condition should be readily recognized and accurately interpreted by the attending physician. Vomiting, of course, is the chief and the outstanding symptom, and, unlike that associated with congenital pyloric stenosis, it begins almost immediately after birth, is progressive and persistent and almost always contains bile. The lower part of the abdomen is relatively flat; the epigastrium is prominent, and often marked peristaltic waves may be seen. The diagnosis, however, in the last analysis really depends on the roentgen observations, inasmuch as with a plain roentgenogram the distended stomach and duodenum can be clearly visualized and no gas is observed lower in the bowel. If a little barium sulfate is given by mouth, the exact lower limit of the obstructed duodenum may be clearly seen.

*Treatment.*—A successful outcome in a case of congenital obstruction of the duodenum demands a very early diagnosis and immediate accurate surgical intervention. Infants fortunate enough to have only a partial obstruction (stenosis) may live for days or months in fair health; those with a complete blocking of the lumen (atresia) die within a very few days. Therefore the necessity for no delay.

It is well to keep in mind that in the preoperative period of preparation the introduction of a Levine tube into the stomach and the administration of adequate amounts of solution of sodium chloride and dextrose (as well as small amounts of blood) will contribute much to the ease of operation and the smoothness of the postoperative course. In the experience of one of us (E. M. M.),[7] which covers a series of 10 cases (with six recoveries), the following technic has been found very satisfactory: The infant (usually weighing in the neighborhood of 5 pounds [2,270 Gm.]) is securely bandaged to a well padded board, and the skin of the entire abdomen and the lower part of the chest is gently but thoroughly prepared with warm soap and water and alcohol. With the area under local anesthesia induced with procaine hydrochloride, combined as a rule with 0.02 grain (0.0012 Gm.) of morphine sulfate administered by hypodermic injection, the abdomen is opened through a right rectus incision, perhaps an inch (2.5 cm.) lower than for a Rammstedt operation. The right lobe of the liver is gently retracted toward the right to expose the dilated duodenum, which at times is almost as large as the stomach itself. One then identifies the position of the transverse colon, the ascending colon and the cecum, determines the presence or

2. Seidlin, S. M.: Congenital Duodenal Septum with Obstruction, Bull. Johns Hopkins Hosp. **37**:328, 1925.

3. Morton, J., and Jones, T. B.: Obstructions About the Mesentery in Infants, Ann. Surg. **104**:864, 1936.

4. Jackson, R.: Congenital Constriction of the Duodenum Due to an Abnormal Fold of the Anterior Mesogastrium, Ann. Surg. **84**:723, 1926.

5. Ladd, W. E.: Congenital Duodenal Obstruction, Surgery **1**:878, 1937.

6. Donovan, E. J., and McIntosh, R.: Disturbances of Rotation of the Intestinal Tract: Clinical Picture Based on Observation in Twenty Cases, Am. J. Dis. Child. **57**:116 (June) 1939.

7. Miller, E. M.: Bowel Obstruction in the Newborn, Ann. Surg. **110**:587, 1939.

absence of a volvulus of the midgut and carefully inspects the field for an abnormal fold or band of adhesions. This procedure may at times involve complete exposure of all of the loops of bowel and may consume several minutes before the exact site and nature of the obstructing mechanism are made clear. Any constricting band is cut across and released, and a volvulus is untwisted; but if an atresia is apparently present or a high grade stenosis exists (it may be difficult to distinguish between the two, especially if the point of obstruction lies hidden beneath the root of the mesentery), nothing but a short-circuiting lateral anastomosis will be effective. Furthermore, it is our belief that when the site of pathologic change is low enough to produce biliary vomiting and the obstruction has caused a pronounced dilatation of the duodenum an anastomosis between the wide duodenum and the collapsed first loop of jejunum (usually antecolic because of the narrow space in the mesentery of the transverse colon between the right and midcolic arteries) is by far the procedure of choice, whereas if the obstruction is high in the duodenum and therefore has not been associated with bile in the vomitus, it calls for a posterior gastroenterostomy. The technical procedure involved in the anastomosis is naturally difficult because of the minuteness of the structures (the collapsed jejunum being about the size of a goose quill), but with great care two rows of the finest silk sutures can be accurately placed (without the use of clamps) and a water-tight anastomosis completed. If perchance there should appear to be a little twisting or narrowing at the proximal end of the suture line a sidetracking enteroenterostomy should be added (as in 1 of our cases) to insure avoidance of a potential dilatation of the afferent loop of the jejunum. It is perhaps needless to mention, but extremely important to emphasize, that the most painstaking and meticulous postoperative care on the part of the surgeon, the pediatrician, the resident and intern staff and the nurses is necessary if a successful result is to be obtained.

### CONGENITAL ATRESIA OF THE ILEUM

In contrast with complete congenital obstruction of the duodenum, the outlook for which is progressively becoming more favorable, atresia of the ileum in a newborn infant has thus far offered a very gloomy prognosis, inasmuch as the number of successful results which have followed operation and later been reported can almost be counted on the fingers of one hand (Fockens,[8] Demmer,[9] Carter[10] and Ladd[11]). As we have intimated in our opening paragraphs, the site of the obstruction is usually low, most often in the region of Meckel's diverticulum, and here, as in the duodenum, may be found a complete gap in the continuity of the bowel (as in the case of the first successful operation, by Fockens, of Rotterdam, in 1911) or a tight narrowing of the lumen sufficient to block the channel effectively. The clinical picture here, as when the duodenum is involved, is unique, but by way of contrast let it be emphasized that the vomiting, which in either type is the chief symptom, begins immediately after birth, is at first composed of stomach contents, then is stained with bile and rapidly becomes brown and has a fetid odor. The lower part of the abdomen, instead of being relatively flat (as with duodenal atresia), becomes progressively distended, and throughout there is

8. Fockens, P.: Ein operativ geheilter Fall von kon. enitaler Dünndarmatresie, Centralbl. f. Chir. **38**:532, 1911.

9. Demmer, F.: Atresiai Ilei. Resectio ileocoecalis; Heilung, Arch. f. klin. Chir. **147**: 471, 1927.

10. Carter, R. F.: Cong. Occlusion of Duodenum and Small Intestine: Two Successful Cases, J. Pediat. **2**:27, 1933.

11. Ladd, W. E.: Personal communication to the author.

a tympanitic note on percussion. The roentgen findings here also are of the greatest importance, because the plain roentgenogram will show clearly the several loops of distended small bowel, and practically no air can be visualized in the colon (fig. 1). Moreover, if a little barium sulfate is given by enema, the collapsed state of the entire large bowel can be easily made out.

*Treatment.*—It must be apparent that nothing save early diagnosis and prompt surgical intervention will be of any avail. Left alone for more than two or three days the rapidly distending completely obstructed small bowel undergoes progressive degenerative changes leading to necrosis, and too often it happens (in fact it is the general rule) that operation is delayed to the point where actual peritonitis has already set in. Moreover, a careful survey of the literature will quickly convince one that an ileostomy alone is of no value, for there has never been reported a permanently favorable result following this procedure. (One of us [E. M. M.]

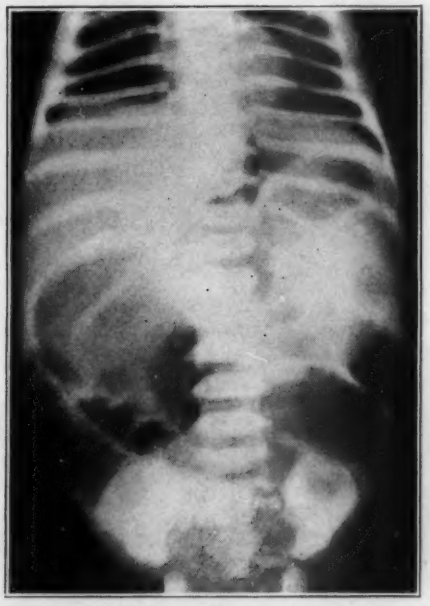

Fig. 1 (case 2).—Roentgenogram showing the distended loops of obstructed small intestine.

and Dr. A. H. Parmelee [12] reported in 1927 the case of an infant living for seven weeks after ileostomy.) In fact, nothing short of an adequate exposure of the site of obstruction and a short-circuiting anastomosis offers the slightest chance of recovery. By presenting here the details of the 2 cases in which we have operated successfully we can perhaps best illustrate the technical points involved in the operations.

## REPORT OF CASES

Case 1.—Infant D. B., a white boy, was born March 22, 1941 by normal delivery. His weight at birth was 6 pounds 12 ounces (3,062 Gm.). He was admitted in an incubator from the South Chicago Community Hospital to Cook County Hospital at 6:30 p. m. March 24, to the pediatric service of Dr. Greengard and Dr. Raycraft (Dr. Irma McFadden, resident). Since birth the infant had been vomiting and during the preceding forty-eight hours he had lost 12½ ounces (354 Gm.). At first the vomitus had been bile stained, but at the time of exami-

12. Miller, E. M., and Parmelee, A. H.: Congenital Bowel Obstruction, S. Clin. North America **7**:649, 1927.

nation it had a brown color and a fetid odor. On examination, uniform distention of the entire abdomen was noted, and with a stethoscope could be heard active peristaltic sounds. The weight on admission was 5 pounds 2 ounces (2,325 Gm.). A roentgengram of the abdomen showed several distended loops of small bowel and no visible gas in the colon. A little of a thin mixture of barium sulfate was then given by enema, and the collapsed state of the large bowel was clearly seen. Since the presence of a congenital obstruction of the small bowel was suspected, surgical consultation was at once obtained, after which preparations were immediately made for operation. A Levine tube was introduced into the stomach, and intravenous administration of solution of sodium chloride and dextrose was started after the vein at the internal malleolus had been cut down on. The operation (by E. M. M.) began

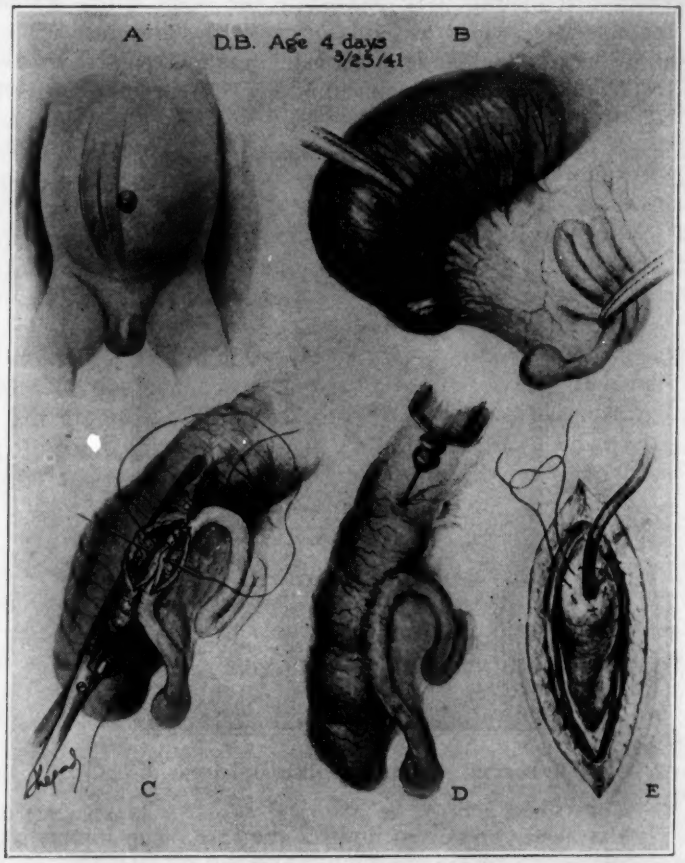


Fig. 2 (case 1).—Steps in the operative correction of atresia of the midportion of the ileum. *A*, right midrectus incision made with the area under local anesthesia; *B*, atresia of the ileum with a gap about ¾ inch (1.9 cm.) long in the continuity of the bowel; *C*, lateral anastomosis; *D*, decompression of the proximal (distended) portion of the bowel with a large aspirating needle; *E*, temporary ileostomy with a no. 10 catheter inserted through the opening made by the aspirating needle.

at 3 p. m., March 25 (weight of the infant 4 pounds 14 ounces [2,210 Gm.]). With the patient under anesthesia induced with procaine hydrochloride, the abdomen was opened through a 3 inch (7.5 cm.) right rectus incision (center opposite the umbilicus [fig. 2*A*]). The distended loops of small bowel were allowed to come out of the abdomen; thus was quickly revealed the atresia of the lower part of the ileum with a complete gap 0.75 inch (1.9 cm.) long in the continuity of the bowel (fig. 2*B*). The proximal (dilated) portion of the bowel was somewhat purplish and measured about 1.5 inches (3.8 cm.) in diameter; the distal (collapsed) portion of the bowel was pale and had a diameter of about 3 mm. The distended

Fig. 3 (case 1).—Roentgenogram twelve hours after ingestion of a barium sulfate test meal. This gastrointestinal study made ten weeks after operation fails to show any lagging of the barium at the site of the anastomosis.

Fig. 4 (case 1).—Appearance of the patient about two years after operation.

part of the bowel was carefully decompressed (care being taken not to soil the surrounding peritoneum) with a no. 20 aspirating needle and syringe (fig. 2*D*), after which a lateral anastomosis (fig. 2*C*) was made with use of 5 0 silk and the smallest needles available. Two rows of sutures were carefully placed (a clamp being used on the proximal portion of the bowel only), and after this a no. 10 catheter was introduced into the proximal segment through the hole made by the aspirating needle to serve as a temporary ileostomy for the escape of gas and thus minimize the tension on the suture line during the period of healing (fig. 2*E*). For closing the incision a running suture of catgut was used for the peritoneum and interrupted sutures of fine silk were used for the sheath of the rectus muscle and the skin.

The postoperative course was surprisingly smooth. The Levine tube was removed after two days, and small amounts of water were given by mouth, parenteral administration of fluids being continued, however, for about one week. Some dark brown material was passed rectally on the second day, and on the fourth day there were three greenish mucous stools. The ileostomy catheter functioned beautifully and after ten days was taken out. Some redness of the skin about the opening was noticed for a while, but as the external drainage became less and less this gradually disappeared. At the close of the third week the wound was entirely healed, and on the thirty-eighth postoperative day the infant left the hospital (May 4), taking his feedings well, having normal bowel movements and showing a gain of approximately 3 pounds (1,360 Gm.). A careful follow-up has revealed most gratifying progress. A slight anemia was observed for several months, and during this time a trace

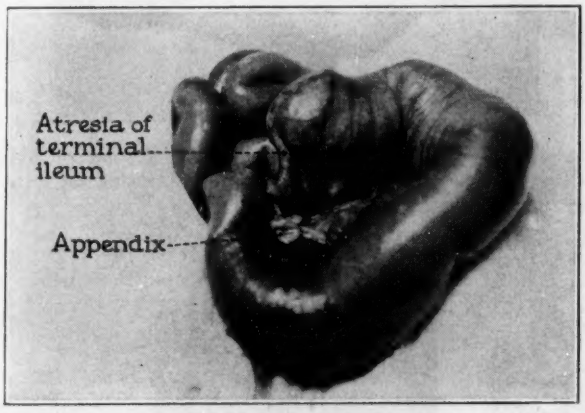


Fig. 5 (case 2).—Photograph made during operation. The anastomosis was made proximal to the ileocecal valve.

of blood was constantly found in the stools, but after one transfusion of blood this entirely disappeared. On June 5, approximately ten weeks after the operation, he was given a little barium sulfate by mouth and a twenty-four hour gastrointestinal study made. This failed to reveal by roentgenogram any lagging in the passage of the meal or even to furnish any clue as to the site of the anastomosis (fig. 3). A photograph of this boy taken on Jan. 5, 1943 (twenty-two months after the operation) shows a normal, healthy child (fig. 4).

CASE 2.—Infant T. C., a white boy, was admitted from the Walther Memorial Hospital to Cook County Hospital at 7:30 p. m. Jan. 12, 1942, to the service of Dr. Greengard and Dr. Raycraft (Dr. Irma McFadden, resident). The baby was born on January 8 at full term, weighing 8 pounds 1½ ounces (3,671 Gm.). He had begun vomiting a bile-stained material immediately after birth, and no meconium had been passed. An enema given on the second day was without result. Administration of 10 minims (0.6 cc.) of castor oil had been followed by a large emesis. Abdominal distention was observed on the third day, and a roentgenogram taken on January 12 revealed several loops of distended bowel. The vomitus by that time had become brown instead of bile colored, and the presence of a congenital obstruction of the small bowel was strongly suspected. On the boy's admission to the Cook County Hospital, on the fourth day, all signs seemed to confirm this impression. A new roentgenogram of the abdomen (fig. 1) clearly showed greatly dilated loops of ileum; the entire abdomen was distended and tense; only faint bowel sounds could be heard, and nothing had been discharged per rectum. A Levine tube was immediately introduced and the administration of

intravenous fluids was begun. At operation (by E. M. M.), on January 13, with the area under local anesthesia, the abdomen was opened through a right midrectus incision. The many distended loops of small bowel were allowed to come out of the abdomen, which allowed the operator to see quickly the point of atresia of the terminal portion of the ileum 2 inches (5 cm.) above the ileocecal valve. Proximal to this site the dilated small bowel measured 2 inches (5 cm.) in diameter; its smooth glistening peritoneal covering had already become dull and gray and was covered with a plastic exudate (fig. 5). The short segment of ileum distal to the atresia was the size of a small angleworm. Deflation of the proximal portion of the bowel was effectively carried out as described for case 1, after which a short-circuiting anastomosis was made (enteroenterostomy). No clamps were used; the suture material was the finest obtainable, and a no. 10 catheter was inserted at the site of needle puncture to serve as an ileostomy. Closure was made as previously described.

The postoperative course in this case, as in case 1, was unexpectedly uneventful. Good use was made of the oxygen tent. Fluids and blood were introduced by vein for about one week. The Levine tube came out on the third day, and feedings by mouth were begun. The ileostomy catheter functioned well and after seven days was removed. Two weeks later the abdominal incision was entirely healed and clean. The gain in weight since then has been steady and uninterrupted. On discharge from the hospital, on March 13, just one month after the operation, the infant weighed approximately 7 pounds (3,175 Gm.) and on Jan. 22, 1943, almost one year after surgical intervention, he weighed 23 pounds (10,433 Gm.). A photograph made on that date revealed a strong, healthy, well nourished normal boy.

## COMMENT

Progress in the surgical correction of congenital obstruction of the duodenum and of the ileum can be made only by close observation of the newborn infant during the first few days of life. Vomiting should not be considered as necessarily due to improper feeding. If it is persistent and remains constantly biliary, it is quite likely due to congenital obstruction of the duodenum. If vomiting persists and the material becomes brown and is associated with a progressive distention of the abdomen, there is in all probability a congenital obstruction of the lower portion of the ileum. In either case a roentgenogram of the abdomen may at once settle the diagnosis, and if it is followed by immediate accurate surgical measures there is a very fair chance of an excellent result.

700 North Michigan Avenue.
4753 Broadway.
1011 Lake Street, Oak Park, Ill.

# Progress in Pediatrics

## POSTOPERATIVE PULMONARY COLLAPSE IN CHILDHOOD

CAPTAIN CLEMENT J. MOLONY
MEDICAL CORPS, ARMY OF THE UNITED STATES

Reports on postoperative atelectasis have been fairly frequent in the last few years, especially since this condition has been recognized as the most common pulmonary complication following a surgical operation. In spite of this, there has never been an inclusive report from a children's hospital. It is thought that such a report might be instructive and at the same time might clarify the problem and its treatment in the pediatric age group.

In order to obtain as large a number of cases as possible and because I have been a resident physician at two large children's hospitals, this report will consider postoperative collapse as seen at the Children's Memorial Hospital in Chicago and the Los Angeles Children's Hospital in the ten year period ending in 1941. Methods of treatment were about the same at both institutions except for one factor: At the Children's Memorial Hospital bronchoscopy is a vital part of the hospital team play, and in about one third of the cases bronchoscopy was performed as part of the treatment.

It is not proposed to present anything new about postoperative collapse. The intention is rather to relate experiences with it in children and perhaps stimulate interest in its proper diagnosis, prophylaxis, course, sequelae and treatment. There will be some discussion of its etiology, so that efforts at treatment will have some foundation in fairly well established fact.

Atelectasis may be defined as an airless state of the lungs; originally coined to refer to the variety found in newborn infants, the word now commonly denotes any collapse of the alveoli, no matter what the cause. Atelectasis was first described satisfactorily and completely by Gairdner,[1] who in a series of papers from 1850 to 1853 carefully differentiated it from pneumonia both grossly and microscopically. Moreover, he laid down the principle that in most cases, both in adults and in children, it is secondary to bronchial obstruction from secretion. It is extremely interesting to note that this first real description of the condition and its cause is in complete agreement with the most widely accepted views today.

After this early description of atelectasis, it was forgotten until Pasteur took it up in 1890.[2] His tenet that collapse occurred from diaphragmatic paralysis then held sway until the First World War and after, when Bradford [3] demonstrated it following wounds of the chest and Jackson and Lee [4] showed its relationship with plugging

From the Service of Dr. Joseph Brennemann, the Children's Memorial Hospital, Chicago, and the Children's Hospital, Los Angeles.

1. Gairdner, W. T.: On the Pathological States of the Lung Connected with Bronchitis and Bronchial Obstruction, Monthly J. M. Sc. **11**:122 and 230, 1850; **12**:440, 1851; **13**:238, 1851.

2. Pasteur, W.: Respiratory Paralysis After Diphtheria as a Cause of Pulmonary Complications with Suggestions as to Treatment, Am. J. M. Sc. **100**:242-257, 1890.

3. Bradford, J. R.: Massive Collapse of Lung, Quart. J. Med. **12**:127, 1919.

4. Lee, W. E.; Tucker, G.; Ravdin, I. S., and Pendergrass, E. P.: Experimental Atelectasis, Arch. Surg. **18**:242-250 (Jan., pt. 2) 1929.

of bronchi by secretions. They postulated a combination of diaphragmatic splinting plus plugs of thick mucus plus absence of cough reflex. Since then a tremendous literature has developed, which has been reviewed elsewhere.[5]

The reports of postoperative atelectasis in children have been meager, and there has never been a report of a series of cases from a children's hospital. Scott[6] reviewed all the literature up to 1925 and found a few cases of postoperative atelectasis in children. King[5] in his report on 200 cases in 1932 included an undetermined number in which the patients were children, probably about 10 in all. Eliason[7] reported 32 cases, in 4 of which the condition occurred in a child. Bader[8] reported the case of a 12 year old boy who recovered spontaneously. Sauer[9] and Levy[10] each reported 1 case; the patients were boys 5 and 14 years of age respectively. Brock[11] reported the cases of 4 children, 1 of whom died on the third postoperative day; at autopsy atelectasis was considered the cause of death.

The general incidence of collapse as a postoperative complication has been changing rapidly as the condition is better understood and looked for. In most large hospitals where pneumonia formerly was the common diagnosis in thoracic complications following an operation, now atelectasis is the more common by a ratio of 3 to 1.

In a children's hospital about the same ratio must exist. There is much evidence to support the idea that almost all so-called postoperative pneumonias are secondary to an initial collapse. In a children's hospital there is a much smaller incidence of postoperative collapse than in a hospital for adults because surgical intervention in the upper part of the abdomen is not frequent and because children are not kept as immobilized and narcotized as are their elders.

The diagnosis of postoperative atelectasis is based on the rather sudden onset of cough, fever and dyspnea in a child who has been operated on a day or so before. There are suppression of breath sounds, dulness, usually rales and usually signs of consolidation over the involved area of the lung, and by its sounds and appearance the heart seems to be shifted to the involved side. The roentgenogram shows a shadow corresponding to a collapsed lobe or to collapsed lobes; the leaf of the diaphragm is high on the involved side, and usually the heart is shifted toward the side of the collapse. Collapse of the upper lobe may or may not pull the heart toward the lesion.

## MATERIALS

The table gives at a glance the data for the whole group of cases and a rough summary of them. All the cases occurred in the ten year period 1931 to 1941.

5. King, D. S.: Postoperative Pulmonary Complications: Statistical Study Based on Two Years' Personal Observation, Surg., Gynec. & Obst. **56**:43-50 (Jan.) 1933. Bowen, D. R.: Acute Massive Collapse (Atelectasis) of Lung, Am. J. Roentgenol. **21**:101-141 (Feb.) 1929. Haight, C., and Ransom, H. K.: Observations on Prevention and Treatment of Postoperative Atelectasis and Bronchopneumonia, Ann. Surg. **114**:243-262 (Aug.) 1941.

6. Scott, W. J. M., and Cutler, E. C.: Postoperative Massive Atelectasis: Effect of Hyperventilation with Carbon Dioxide, J. A. M. A. **90**:1759-1763 (June 2) 1928.

7. Eliason, E. L., and McLaughlin, C. W., Jr.: Postoperative Pulmonary Atelectasis, S. Clin. North America **14**:1-12 (Feb.) 1934.

8. Bader, G. B.: Postoperative Massive Atelectasis of Lung: Case Report, J. Pediat. **2**:726-728 (June) 1933.

9. Sauer, L.: Postoperative Atelectasis: Report of Case, Promptly Relieved by Simple Measures, J. Pediat. **11**:321-323 (Sept.) 1937.

10. Levy, H., and Litvak, A. M.: Postoperative Pulmonary Atelectasis, J. Pediat. **10**:632-640 (May) 1937.

11. Brock, R. C.: Post-Operative Chest Complications: Clinical Study, Guy's Hosp. Rep. **86**:191-247 (Jan.-April) 1936.

I. *Sex.*—In this series the sexes were about equally represented. It is interesting that in all reports from hospitals for adults it is stated that postoperative pulmonary collapse occurs about three times as often in males as in females. The explanation is that a man breathes abdominally while a woman uses her intercostal muscles to a greater extent. Since the abdomen is painful after surgical intervention, a man tends to splint his breathing, and deficient expansion of the lungs results in collapse.

*Summary of Data on 21 Cases of Postoperative Pulmonary Collapse in Children*

| Case and Sex | Age | Operation | Anesthetic | Temperature with Collapse, F. | Involved Lobe | Day Occurred | Bronchoscopy, Days after Collapse | Day Cleared |
|---|---|---|---|---|---|---|---|---|
| 1 M | 5 | Nephrectomy | Ethylene | 103.2 | RL | 3d | 3d and 7th | Bronchoscopy, 10 |
| 2 F | 10 | Intestinal obstruction | Nitrous oxide | 105 | RU | 2d | ..... | Spontaneous, 14 |
| 3 M | 4 | Tonsillectomy | Ether | 104 | RM | 2d | ..... | Spontaneous, 9 |
| 4 M | 9 | Appendectomy | Nitrous oxide | 103 | LL | 5th | 2d | Bronchoscopy, 5 |
| 5 M | 9 | Appendectomy | Ether | 102 | LL | 4th | ..... | Spontaneous, 10 |
| 6 F | 1 | Intussusception | Ether | 104 | RU | 2d | 10th | Bronchoscopy, 13 |
| 7 F | 9 | Appendectomy | Ethylene | 106 | LL | 2d | ..... | Spontaneous, 9 |
| 8 F | 11 | Appendectomy | Ethylene | 103.8 | LL | 2d | ..... | Spontaneous, 5 |
| 9 F | 6 | Intestinal obstruction | Ethylene | 101 | RU | 4th | ..... | Spontaneous, 5 |
| 10 F | 6 | Appendectomy | Ethylene | 104.4 | RU and RL | 3d | ..... | Spontaneous, 7 |
| 11 F | 9 | Tonsillectomy | Ether | 104.6 | LU | 1st | ..... | Spontaneous, 7 |
| 12 F | 11 | Appendectomy | Nitrous oxide | 102.4 | RL | 5th | ..... | Spontaneous, 11 |
| 13 F | 7 | Herniorrhaphy | Ethylene | 106 | RU | 1st | ..... | Spontaneous, 7 |
| 14 M | 1 | Adenoidectomy | Ether | 104.2 | RL | 1st | 2d | Bronchoscopy, 3 |
| 15 M | 5 | Tonsillectomy | Ether | 104 | RU | 3d | 2d | Bronchoscopy, 8 |
| 16 M | 7 | Appendectomy | Ether | 105 | LL and RL | 2d | ..... | Spontaneous, 3 |
| 17 M | 10 | Appendectomy | Ether | 104 | LL and RL | 2d | ..... | Spontaneous, 2 |
| 18 F | 9 | Appendectomy | Ether | 105 | LL | 2d | ..... | Spontaneous, 4 |
| 19 F | 8 | Appendectomy | Ether | 104.6 | RL and RU | 2d | ..... | Spontaneous, 20 |
| 20 M | 3 | Herniorrhaphy | Ether | 104 | RL and RU | 1st | ..... | Spontaneous, 10 |
| 21 M | 8 | Appendectomy | Ether | 103 | RL | 3d | ..... | Spontaneous, 5 |
| M, 10<br>F, 11 | 0- 4:4<br>5- 8:8<br>9-12:9 | Abdominal, 16<br>Others, 5 | Ether....12<br>Ethylene 6<br>Nitrous oxide.... 3 | 104+, 13<br>102+, 7<br>101+, 1 | RL, 9<br>LL, 7<br>RU, 7<br>LU, 1<br>RM, 1 | 1st, 5<br>2d, 8<br>3d, 4<br>4th, 2<br>5th, 2 | Average, 4th | Spontaneous, 9<br>Bronchoscopy, 8 |

II. *Age.*—The age of the patients in this group of cases tends to fall in the range of older children. This probably is accounted for by the number of patients operated on and has no significance.

III. *Type of Operation.*—In 15 of the cases, including 2 herniorrhaphies, collapse followed operation on the abdomen; in 4 it followed tonsillectomy and adenoidectomy, and in only 1 did it follow another type of operation, a nephrectomy which was performed via the retroperitoneal route but necessitated a huge incision.

This series fits in nicely with others from other hospitals. Abdominal operations, especially those on the upper part of the abdomen, produce a great amount of postoperative pain, with resulting splinting of respiration. The lungs are not expanded properly, and the stage is thus set for collapse.

IV. *Anesthetic.*—Ether administered by the open mask method was the most common anesthetic used. However, when it is explained that at one of the hospitals (Los Angeles Children's Hospital) this is the only anesthetic used, this general figure is not completely valid. At the other hospital open ether anesthesia and the closed method of anesthesia using nitrous oxide and ethylene were employed in about equal numbers of cases for abdominal operations. In the group from that hospital, in 7 cases collapse followed the closed bag method and in 8 cases it followed open ether anesthesia. If the 4 cases in which collapse followed tonsillectomy are eliminated and only those considered in which it followed an abdominal operation, then in 7 of 11 cases (64 per cent) it occurred after a closed bag type of anesthesia. This is interesting because recently it has been shown that the closed bag method of anesthesia may result in displacement of nondiffusible alveolar air by oxygen and the anesthetic gases. Both of these are absorbed by the blood stream in a matter of a few minutes, and collapse may occur right at the time of operation. In this series there is no correlation between the anesthetic used and the time the atelectasis appeared.

V. *Symptoms.*—As is seen, the rise of temperature with collapse was marked, in the majority of cases reaching 104 F. or more. The symptoms were definite in most cases: high fever, an average pulse rate of 130 per minute and an average respiratory rate of 34 per minute. The majority of children showed dulness to percussion, suppression of breath sounds, some bronchophony and sticky rales. In 13 (62 per cent) the heart was shifted clinically toward the side of the atelectasis. Cyanosis was present in only 5 cases. In general, the patients were acutely ill, with sufficiently definite symptoms to allow a diagnosis to be made. All lobes of the lung were involved, with the lower lobes predominating. Collapse occurred most often in the lower lobe of the right lung. The bronchus to this lobe is anatomically straight on from the trachea and is the favorite seat of atelectasis because thick secretions gravitate to this dependent point.[12] In general, small babies tend to have the obstruction in the bronchus leading to an upper lobe; this was not found in our series.

V. *Day of Occurrence.*—In all cases collapse had occurred by the fifth postoperative day, with the second day being the average for the group. Actual beginning collapse must occur sooner than this, but until the process includes infection the patient may not show enough symptoms and signs to make one suspect a pulmonary complication.

VII. *Day of Clearing.*—In the cases in which the condition cleared spontaneously, it did so on an average about the ninth day. In the 5 cases in which treatment was by bronchoscopic drainage the condition cleared on the eighth day. This difference is not significant, of course, when the number of cases is so small. However, the latter group included some of the sickest children, and in case 16 bronchoscopy was done late because the pulmonary conditions were not clearing up spontaneously. In case 21 clearing took a long time, and this child later acquired severe glandular tuberculosis.

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12. Coryllos, P. N., and Birnbaum, G. L.: Lobar Pneumonia Considered as Pneumococcic Lobar Atelectasis of Lung: Bronchoscopic Investigation, Arch. Surg. **18**:190-241 (Jan., pt. 2) 1929.

## REPORT OF CASES

CASE 1.—G. O., a boy 5 years of age, was admitted to the Children's Memorial Hospital on July 8, 1937, with a history of gradual enlargement of the abdomen since birth. Examination showed a large cystic mass in the left side of the abdomen. Intravenous pyelograms showed the outline of a large hydronephrosis on the left. Nephrectomy was performed on July 13, 1937. Preoperative medication was ½ grain (0.03 Gm.) of codeine [12a] and 1/300 grain (0.2 mg.) of atropine. Ethylene and oxygen supplemented with ether was used, and the

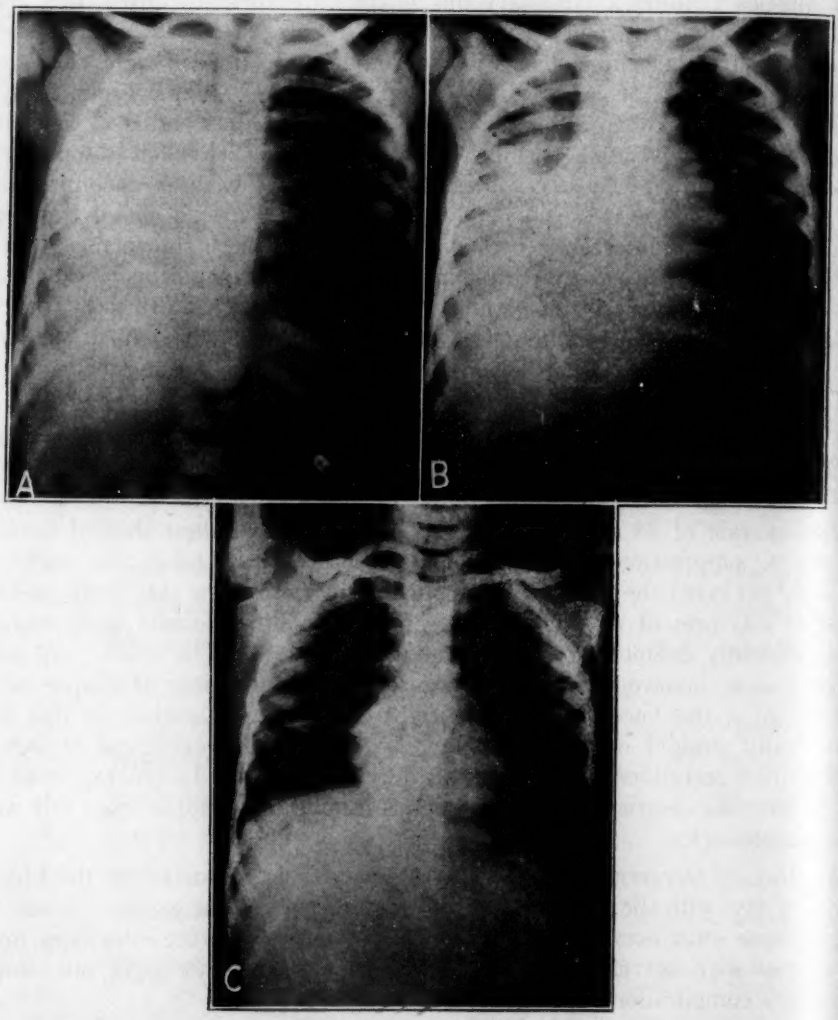


Fig. 1 (case 1).—*A*, roentgenogram taken three days after operation; note the complete shift of the mediastinum. *B*, two days later, after bronchoscopic drainage. *C*, on the patient's discharge from the hospital.

duration of anesthesia was seventy-five minutes. The postoperative course was essentially uneventful until seventy hours later, when the temperature went to 103.2 F., the pulse rate to 140 and the respiratory rate to 34. There were suppression of breath sounds, dulness, bronchophony and crepitant rales over the entire right side of the chest. The heart was shifted to the right. A roentgenogram showed a massive collapse of the right lung (fig. 1 *A*). As the child was not severely distressed, conservative measures were carried out. There was no improvement, and bronchoscopy was done two days later. A large amount of tena-

12a. In all cases in which codeine or atropine was given the sulfate salt was the drug used.

cious purulent material was aspirated from the right main bronchus. There was some improvement in the child's general condition, and some clearing was noted on physical and roentgen examination (fig. 1 *B*). Bronchoscopy was repeated four and seventeen days later. After the second bronchoscopy the temperature dropped to normal. After the third bronchoscopy there was complete clearing (fig. 1 *C*), and physical examination revealed no abnormalities except occasional rales over the lower left side of the chest. The patient's father had asthma, and this child has recently had several mild asthmatic attacks. There has been no apparent residual pulmonic lesion two years after the collapse. This patient has been reported on previously. The relationship of the asthma and the postoperative atelectasis may be of importance.

CASE 2.—H. L., a 10 year old girl, was admitted to the hospital on April 11, 1938, with a history of abdominal pain, vomiting and fever for forty-eight hours. Important in her past history was an appendectomy eight months before. The child was seriously dehydrated and acutely ill. The abdomen was not distended. There was no point of tenderness. About every five minutes loud borborygmi were heard, and at this time the child would wince with pain. The diagnosis of mechanical obstruction of the bowel was made, and exploratory laparotomy revealed a thick, fibrous band encircling the mesentery of the terminal portion

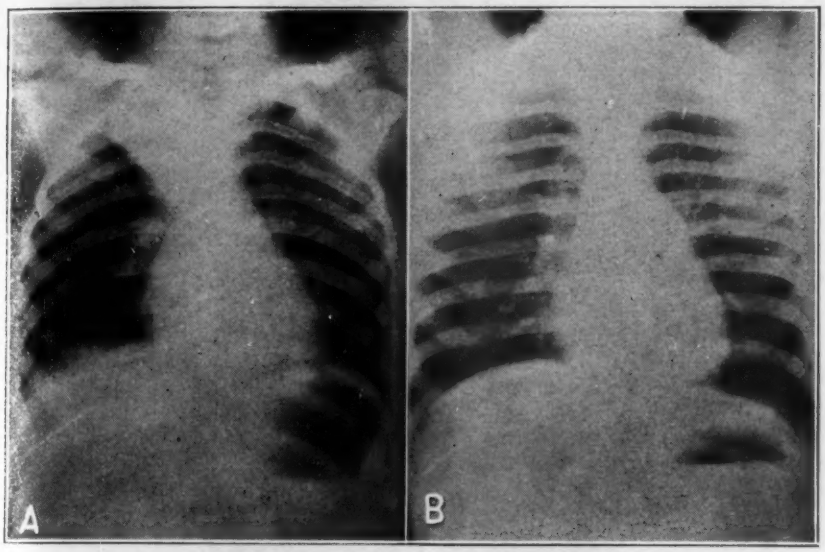


Fig. 2 (case 2).—*A*, complete collapse of the upper lobe of the right lung; *B*, complete clearing four days later.

of the ileum. The band was divided, and the bowel soon assumed a good pink color and filled with gas. Codeine (⅜ grain [0.25 Gm.]) and atropine (1/200 grain [0.3 mg.]) were given preoperatively, and nitrous oxide-oxygen anesthesia was used for a total of forty minutes.

The postoperative course was stormy, and after thirty-six hours the temperature went to 105 F., the pulse rate to 140 and the respiratory rate to 36. There was a moderate dyspnea and cyanosis. Rales were heard throughout the chest, with suggestive bronchial breathing over the lower left lobe of the lung and suppression over the upper right lobe of the lung. A roentgenogram at this time showed nothing unusual. The child's condition improved considerably, although rales continued to be present over the base of the left lung posteriorly and suppression of breath sounds over the upper lobe of the right lung. The temperature came down to normal in about four days. The rales disappeared, and on physical examination the chest was essentially normal except for moderate suppression over the entire right side. The heart seemed to be in normal position. However, a roentgenogram taken on April 21 (fig. 2 *A*) showed a collapse of the upper lobe of the right lung. The child made an uneventful recovery, and a roentgenogram taken April 25, 1938 (fig. 2 *B*) showed complete clearing of the collapsed lobe.

CASE 3.—M. M., a white boy aged 4 years, had a routine tonsillectomy and adenoidectomy performed on Nov. 19, 1934. He was discharged the next day, and two hours after his

return home a fever and cough developed and he complained of abdominal pain. His past history included mild asthma and ptyalism since he was 2 months of age.

The child was readmitted to the hospital November 24. At this time the temperature was 104 F., respirations were rapid and grunting, and there were dulness and suppression of breath sounds over the right side of the chest in the anterior axillary region. The heart tones were loudest to the right of the sternum. Roentgenograms showed moderate, rather diffuse clouding over the region of the middle lobe of the right lung and the heart drawn to the right. The temperature came down to normal on the third day and remained normal.

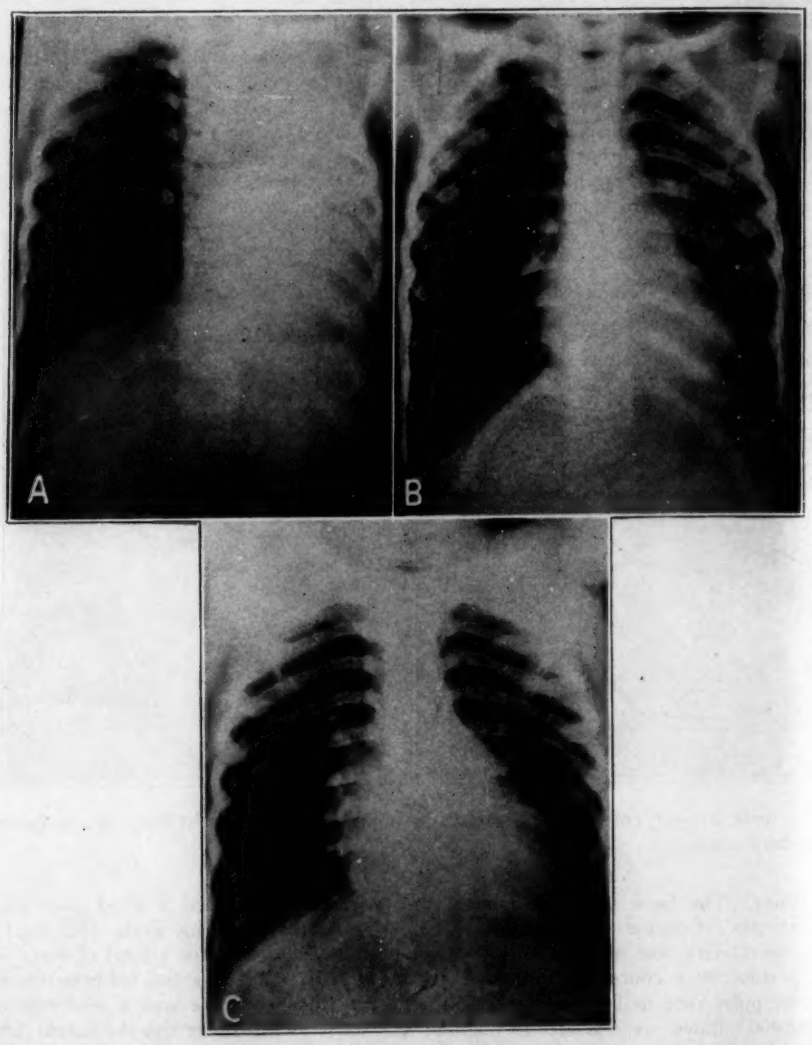


Fig. 3 (case 4).—*A*, complete collapse of the left lung four days after operation; *B*, rapid clearing twenty-four hours later, after the cough had become productive; *C*, clear chest on the patient's discharge from the hospital.

The physical conditions corrected themselves, and roentgenograms taken November 30 showed that the pulmonary fields had almost entirely cleared. The child has been followed in the allergy clinic, and the chest has remained clear. No special treatment was given for his collapse.

CASE 4.—M. D., a 9 year old boy, was admitted to the Children's Memorial Hospital on March 3, 1936 with a history of abdominal pain and vomiting for thirty-six hours. The diagnosis of acute appendicitis was made, and an acute, inflamed, indurated appendix with a

gangrenous tip was removed. Codeine (¼ grain [0.015 Gm.]) and atropine (1/400 grain [0.15 mg.]) were given preoperatively. The anesthetic was nitrous oxide and oxygen with ether and the time thirty minutes. On March 8, four and a half days after the operation, the child's temperature went to 103 F., with a respiratory rate of 36 and a pulse rate of 120. There were a slight amount of mucoid sputum and moderate dyspnea and cyanosis. The left side of the chest appeared immobile, while the cardiac apical impulse was visible in the anterior axillary line. The percussion note was flat over the entire left side of the chest, and there was bronchial breathing over this area. A roentgenogram showed a massive collapse of the left lung (fig. 3*A*). Inhalations of carbon dioxide (20 per cent) and oxygen (80 per cent) were given hourly, and the position was changed frequently. The amount of sputum increased greatly after the inhalations, and in about five hours breath sounds came through almost normally over the left side of the chest. The heart was almost back to normal position, and the percussion note was resonant. Roentgen examination the next day (fig. 3*B*) showed almost complete clearing. Bronchial breathing and bronchophony over the left base persisted, and so bronchoscopy was performed on March 10. The entire bronchial tree appeared red, and there was about an equal amount of pus aspirated from both sides. After this there was almost complete clearing except for occasional rales over the lower part of the left side of

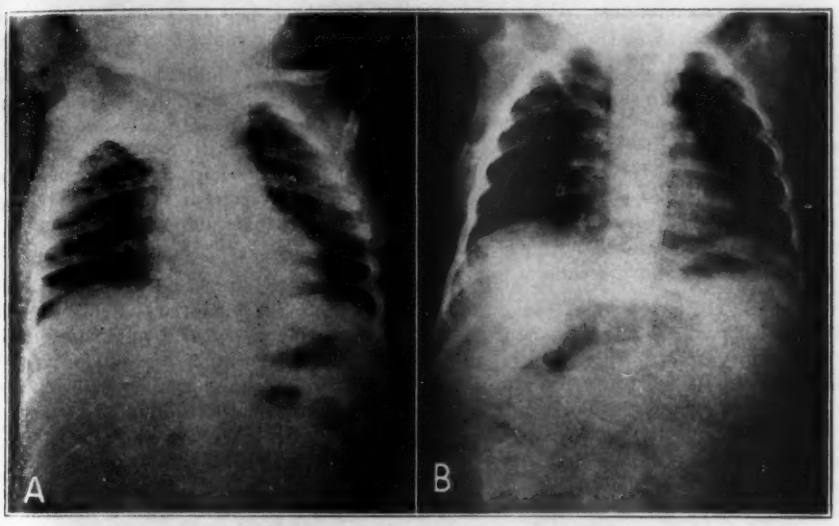


Fig. 4 (case 6).—*A*, upper lobe of the right lung dense two days after operation and the mediastinum not shifted; *B*, chest clearing three days later.

the chest. Roentgenograms taken one month later (fig. 3*C*) and three months later showed the lungs to be normal.

Case 5.—R. K., a boy 9 years of age, was admitted to the Children's Memorial Hospital on March 31, 1930, with a history of abdominal pain and vomiting of eighteen hours' duration. The abdominal pain was localized in the right lower quadrant. Examination indicated acute appendicitis, and an acutely inflamed appendix was removed at operation. Atropine (1/200 grain [0.2 mg.]) was given preoperatively. The anesthetic used was ether, and the total time of anesthesia was twenty-five minutes. The child's postoperative course was uneventful until seventy-two hours after operation, when the temperature went up to 102 F. and the child began to have a cough productive of a tenacious, mucoid sputum. He complained of pain in the left side, and on physical examination there were dulness, bronchial breathing and crackling rales over the lower lobe of the left lung. The heart seemed somewhat to the left. A roentgenogram showed increased density in the region of the lower lobe of the left lung with the heart pulled to the left. The child continued to cough for about one week, and there was a daily elevation of temperature; then there was gradual clearance, shown both by physical and by roentgen examination. He received no special treatment for his condition.

Case 6.—M. P., a Negro girl 14 months of age, was admitted to the Children's Memorial Hospital on March 4, 1939, with a history of having awakened at 4:00 a. m. two days before,

screaming as though in pain. She continued to cry periodically and to have colicky pain. In the fifteen hours before admission the infant had passed several loose stools containing much mucus and bright red blood. On physical examination she was extremely listless. The right ear drum was red and full, and the throat was acutely inflamed. There was a visible and palpable soft mass in the left lower quadrant of the abdomen and extending up into the lower part of the left upper quadrant, while rectal examination revealed a cervix-like mass present in the rectum; a gush of blood and mucus followed the removal of the examining finger. The diagnosis of intussusception was made, and the laparotomy showed one of the ileocecal type present. There was no preoperative medication. Ether anesthesia was used, and the duration was fifty minutes. Because of the extent of the necessary incision, a rather tight abdominal binder was applied. The postoperative course was essentially uneventful until forty-eight hours after operation, when the temperature went to 104 F. and bronchial breathing and bronchophony were noted over the upper lobe of the right lung. No shift of the heart was detected. A roentgenogram showed a collapse of the upper lobe of the right lung (fig. 4 *A*). Respirations were rapid and shallow. The tight abdominal binder was removed, and almost immediately the respirations were deeper and much easier. The temperature gradually came down to normal within four days, although signs of consolidation persisted. Bronchoscopy was performed on March 17. The right main bronchus was inflamed, and considerable mucopurulent secretion was aspirated. Three days later the chest was normal to physical examination and a roentgenogram showed almost complete clearing (fig. 4 *B*).

Case 7.—O. S., a 9 year old white girl, was admitted to the Children's Memorial Hospital April 5, 1939, with a history of abdominal pain for eighteen hours. A diagnosis of appendicitis was made, and an acutely inflamed appendix was removed. Twenty-four hours postoperatively her temperature rose to 106 F. and her respiratory rate to 42 per minute. On physical examination there was silence over the lower lobe of the left lung and some bronchial breathing over the upper portion. The heart was definitely somewhat to the left. Conservative treatment was carried out, and the temperature came down to 100 F. in forty-eight hours and remained low. There was gradual improvement, and by April 15 the chest was clear to physical examination and the child was discharged.

Case 8.—B. H., a 10½ year old girl, was admitted to the Children's Memorial Hospital on Feb. 4, 1938, with a diagnosis of acute appendicitis. She also had a mild infection of the upper respiratory tract. Thirty-six hours after surgical intervention the child's temperature suddenly rose to 103.8 F., the respiratory rate to 28 and the pulse rate to 130. There was diminished excursion of the left side of the chest with bronchial breathing and dulness over the lower part of the left side of the chest posteriorly. The heart was shifted to the left so that the apex impulse was about 3 cm. to the left of the nipple line. Roentgenograms showed a decreased areation of the entire left side of the chest with a shift of the heart to the left (fig. 5 *A*). Inhalations of carbon dioxide and oxygen were given, and the child's position was changed frequently. The temperature came down to normal in forty-eight hours. Bronchial breathing and bronchophony persisted at the base of the left lung, but the heart returned to normal position. In roentgenograms taken at this time, February 9 (fig. 5 *B*), a faint homogeneous triangular shadow could be seen through the shadow of the heart and the left side of the diaphragm was elevated. The chest completely cleared in the next two days, and the child was discharged on February 14. Roentgenograms taken two weeks later showed complete recovery (fig. 5 *C*).

Case 9.—L. G., a 6 year old white girl, was admitted to the Children's Memorial Hospital on Dec. 20, 1936, with a history of generalized abdominal pain, vomiting and occasional loose stools for three days. On examination there was a distended abdomen but no tenderness. Because of an infection of the upper respiratory tract with postnasal drip, operation was delayed for forty-eight hours. At operation Meckel's diverticulum was found; its adhesions were obstructing the small bowel. The patient was given ½ grain (0.03 Gm.) of codeine and 1/200 grain (0.3 mg.) of atropine. Ethylene and oxygen anesthesia was used for one hundred and five minutes.

Her postoperative course was stormy, but she gradually became better except for a cough until the fourth postoperative day, when the temperature was elevated, the cough was worse and coarse moist rales were heard over both pulmonary fields. The next day there was suppression of breathing over the upper part of the right side of the chest, and the heart both clinically and by roentgenogram (fig. 6 *A*) was seen to be pulled to the right. She was treated by frequent change of position and was urged to cough. The process cleared spontaneously in five days (fig. 6 *B* and *C*). It is interesting that the child had an infection of the upper respiratory tract at the time of operation, had an unusually long anesthesia and

had rather large doses of codeine (½ grain [0.03 Gm.]) before the operation and three times in the first few hours after the operation.

CASE 10.—M. S., a white girl aged 6 years, was admitted to the hospital on Oct. 14, 1939 because of the passage of large quantities of blood by rectum. Exploratory laparotomy was done on October 21, at which time nothing abnormal was found; the appendix was removed. Preoperative medication was codeine (¼ grain [0.015 Gm.]) and atropine (1/400 grain [0.15 mg.]). Ethylene and oxygen anesthesia was given for a period of ninety-two minutes.

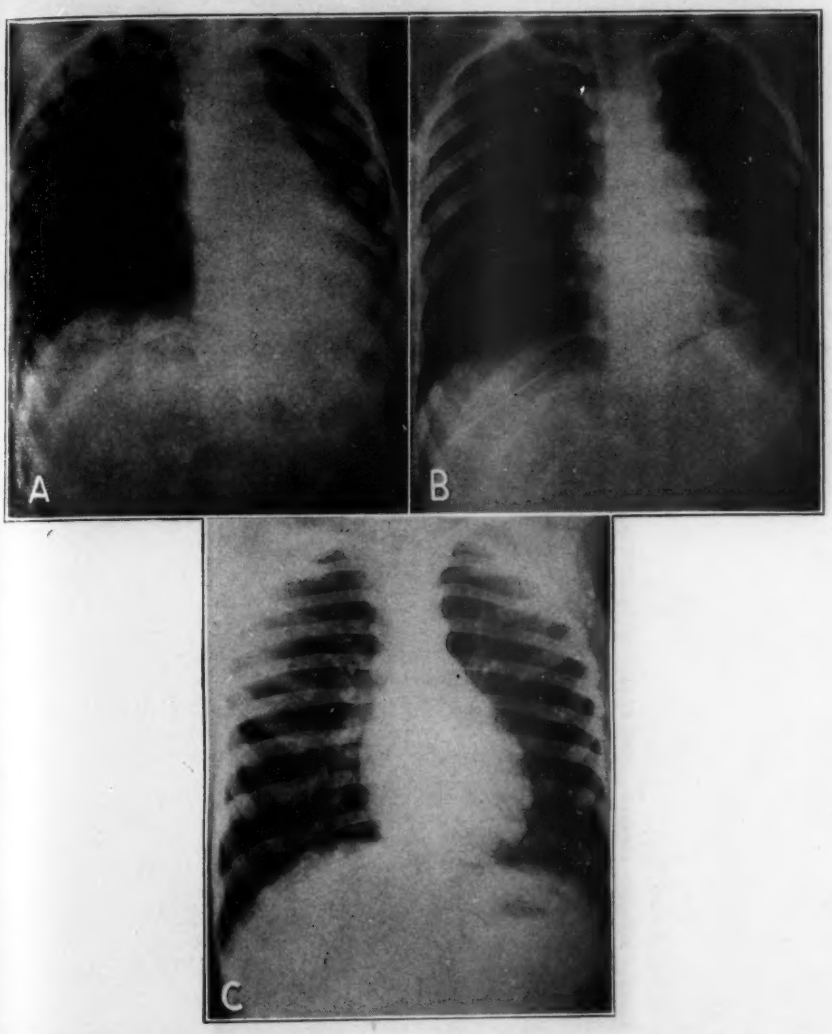


Fig. 5 (case 8).—*A*, collapse of the left lung evidenced thirty-six hours after operation; *B*, collapse still present, as evidenced by the faint triangular shadow behind the heart and the elevation of the left side of the diaphragm; *C*, complete recovery two weeks later.

The postoperative course was uneventful until three days later, when the temperature went to 104.4 F., the pulse rate to 140 and the respiratory rate to 36. There was bronchial breathing over the lower lobe of the right lung, with dulness but no rales. There seemed to be some displacement of the heart to the right. A roentgenogram showed irregular, lobular collapse of the mesial half of the upper lobe of the right lung (fig. 7 *A*). The child was given inhalations of carbon dioxide and oxygen, and her position was changed frequently. The next day the temperature again went to 104.6 F.; then within twenty-four hours it came down to normal and remained there. Coincidentally with the decrease in temperature the child was

much improved and the physical abnormalities were less prominent. A roentgenogram taken on October 28 showed the clouding previously noted at the apex to be cleared, but new areas of collapse were present in the region of the lower lobe of the right lung (fig. 7 *B*). The general clinical course continued uneventful, and roentgenograms taken on October 30 showed almost complete clearing (fig. 7 *C*).

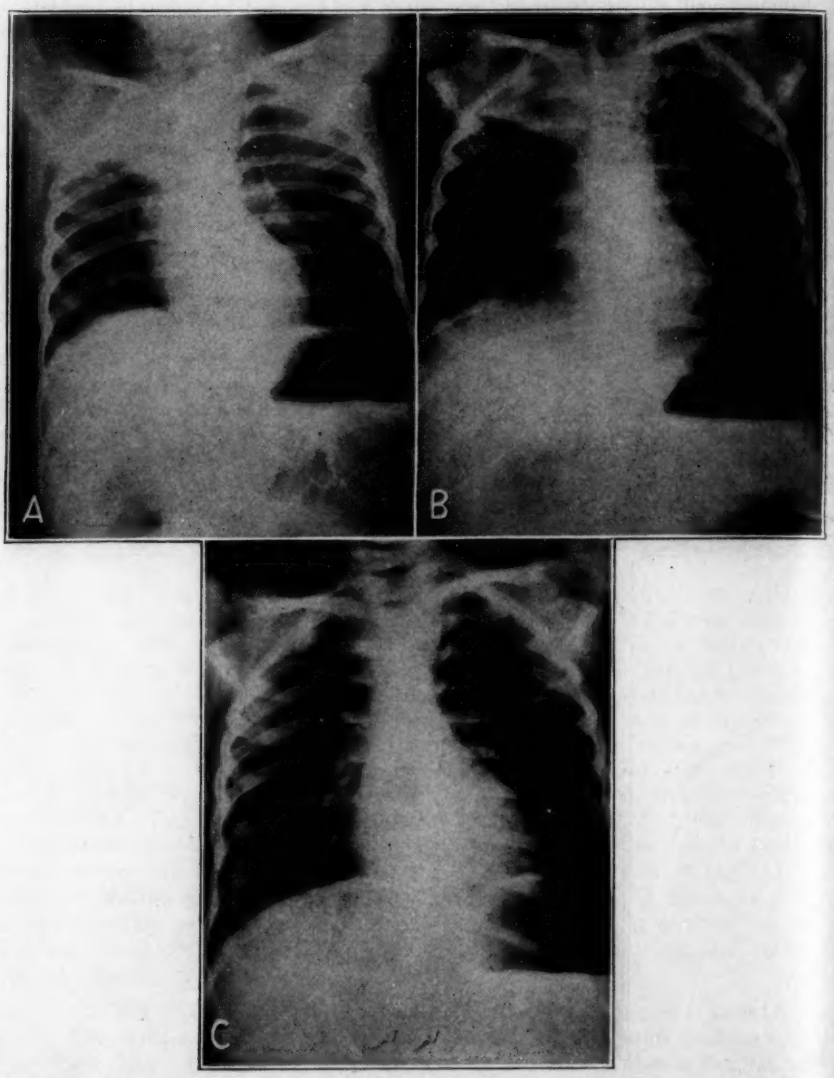


Fig. 6 (case 9).—*A*, condition of the chest one day after the onset of symptoms of collapse (fifth postoperative day), the mediastinum not shifted; *B*, and *C*, clearing process over a period of five days (the chest completely clear in *C*).

CASE 11.—I. S., a 9 year old white girl, had a routine tonsillectomy and adenoidectomy on July 21, 1939 at the Children's Memorial Hospital. A few hours after the child was home, she began to cough and had an elevated temperature. The next day, July 23, pain under the left arm and shoulder developed, and that evening respirations became rapid and the temperature was 103 F. She was readmitted to the hospital on July 24. At this time the temperature was 104.6 F., the pulse rate 134 and the respiratory rate 28. There were dulness, suppression of breath sounds and occasional rales over the upper part of the left side of the

chest anteriorly. Clinically the heart was displaced to the left. A roentgenogram showed diffuse clouding over the lower half of the left pulmonary field and slight displacement of the mediastinal structures to the left. Within three days the temperature dropped to normal, the chest was clear to physical examination and a roentgenogram showed clearing of the previously clouded area.

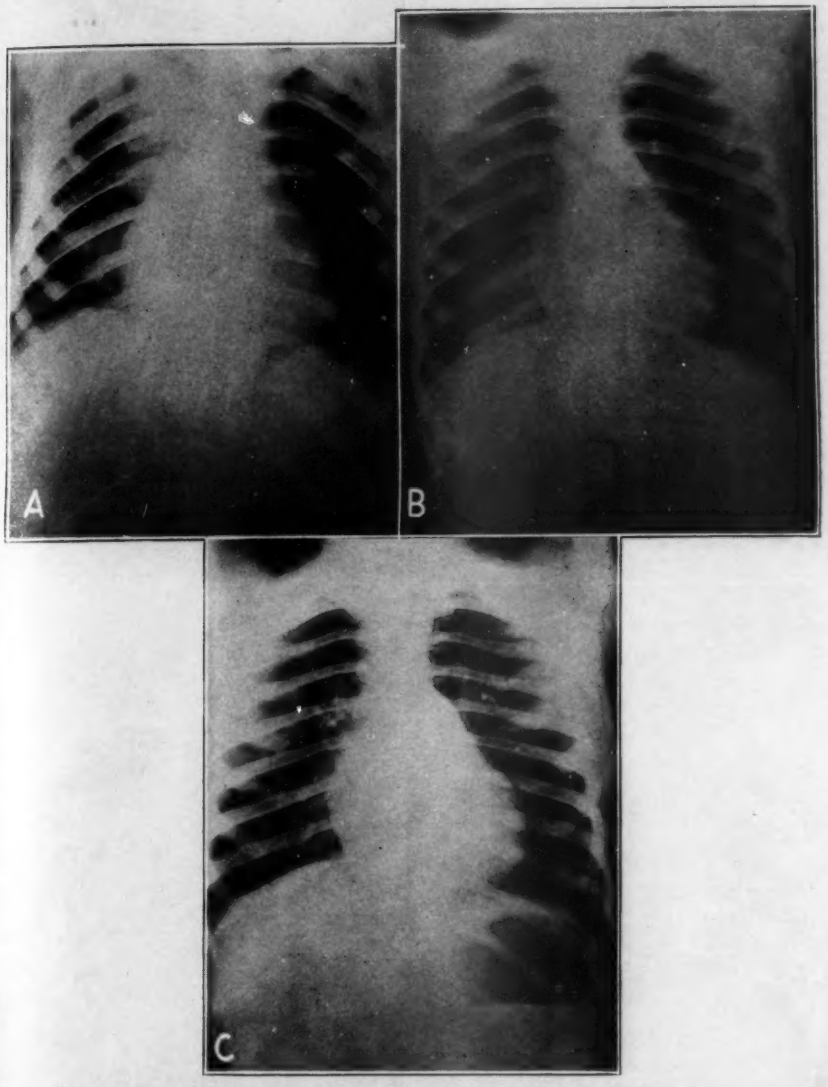


Fig. 7 (case 10).—*A*, collapse of the upper lobe of the right lung three days after operation; *B*, upper lobe clear but a triangular area of collapse at the base of the right lung (seventh postoperative day); *C*, chest clear two days later.

CASE 12.—D. B., an 11 year old white girl, was admitted to the hospital on Oct. 24, 1933, with a history of abdominal pain, vomiting and fever for three days. The child also had congenital syphilis. A diagnosis of acute appendicitis was made, and a gangrenous, perforated appendix was removed. The course was essentially uneventful except for a severe cough with normal conditions in the chest. Atropine (1/500 grain [0.12 mg.]) every six hours for twenty-four hours was prescribed on October 28 because of the extremely wet cough. On October 30 the temperature went to 102.4 F. and the respiratory rate to 42. There were some

suppression of breath sounds, bronchial breathing, bronchophony and rales over the lower lobe of the right lung. A roentgenogram showed partial collapse of the lobe. Conservative treatment was carried out, and there was complete clearing on physical examination by November 6.

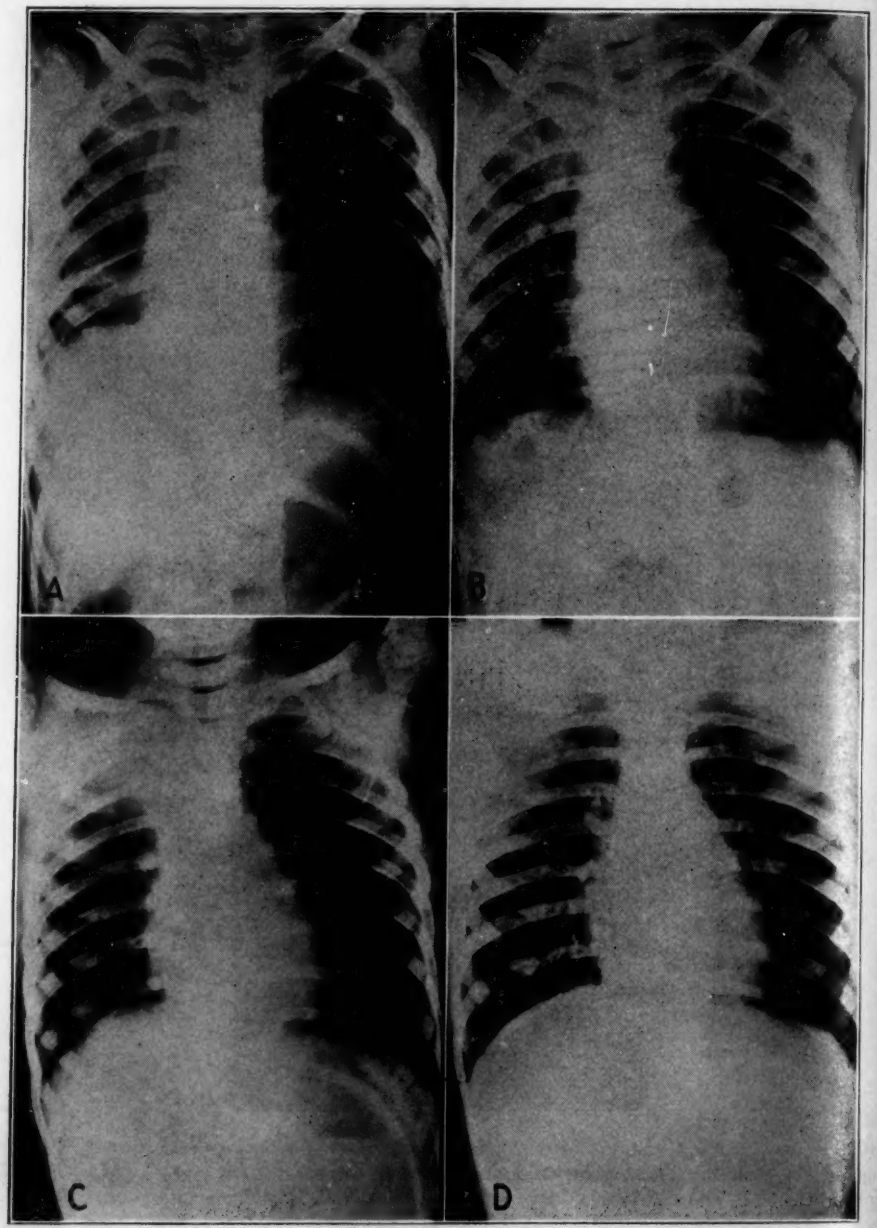


Fig. 8 (case 13).—*A*, beginning collapse of the entire right side twenty-four hours after operation; *B*, the heart has shifted back but some atelectasis persists in the upper lobe of the lung twenty-four hours later; *C*, increased density of the upper lobe of the right lung four days later; *D*, complete clearing four days later.

CASE 13.—A. H., a 7 year old girl, was admitted to the Children's Memorial Hospital on Feb. 6, 1938, for an elective herniorrhaphy. Her temperature was 100.6 F., but on the

day of operation it was normal and there were only 7,600 leukocytes. The following day the temperature had reached 106 F., and there were dulness, bronchial breathing and bronchophony on the right side, with the heart shifted noticeably to the right. Roentgen examination confirmed the diagnosis of collapse on the right side (fig. 8 *A*). Without any particular treatment except frequent changes of position and frequent attempts to raise sputum, the temperature was only slightly elevated the next day and the heart was back in normal position (fig. 8 *B*). Four days later abnormalities persisted in the region of the upper lobe of

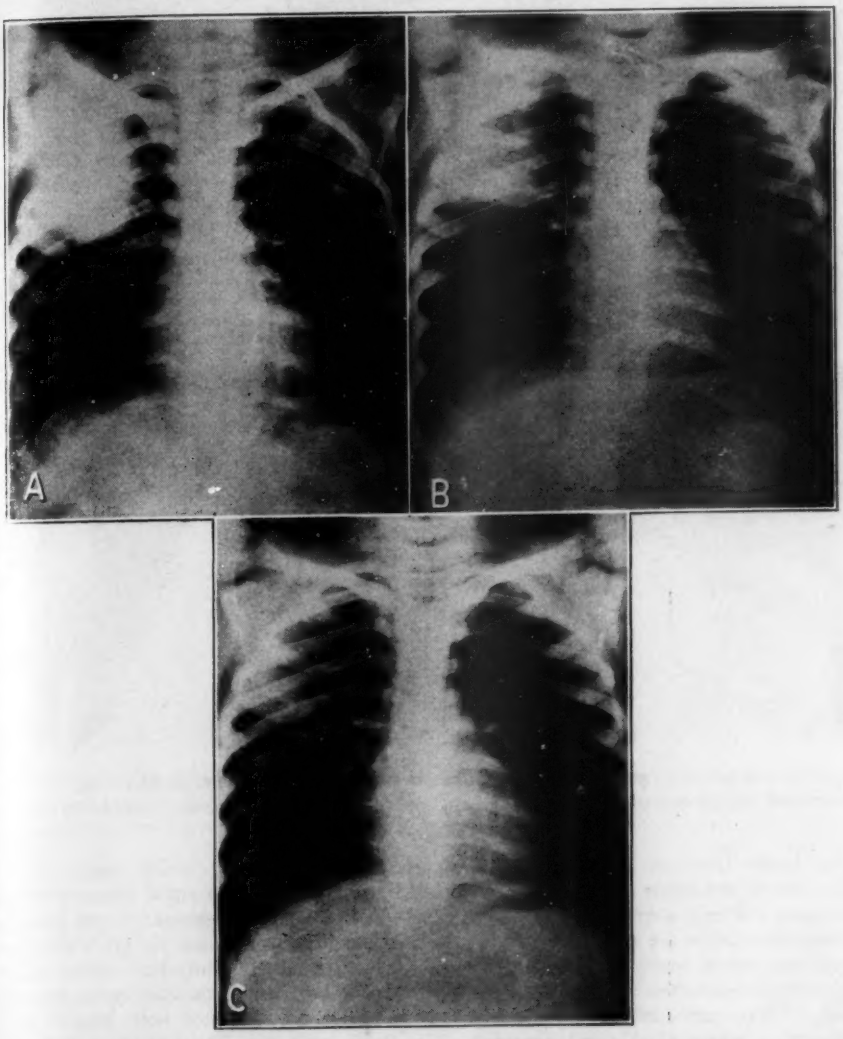


Fig. 9 (case 15)—*A*, roentgenogram of the chest on the seventh postoperative day; *B* and *C*, slow resolution of the process after bronchoscopic drainage of pus from the bronchus of the upper lobe of the right lung.

the right lung, and roentgen examination revealed a persistent and more pronounced atelectasis in that area (fig. 8 *C*). She then became well, and a roentgenogram four days later showed a normal chest (fig. 8 *D*). This series of roentgenograms shows remarkably well the course of an atelectasis from the early beginning collapse of the whole lung through the phase of reexpansion of most of it to the final density of the lobe which is to bear the complete collapse.

Case 14.—B. S., a 13 month old boy, was admitted to the Children's Memorial Hospital on Nov. 23, 1936, for an adenoidectomy. His history included slow development after cyanosis

for a few days after birth, many colds and a chronic watery discharge from the nose. Open ether anesthesia was used, and no preoperative medication was given. The afternoon of the operation the temperature rose to 104 F. and there were bronchial breathing in the right axilla and some dulness over the right midportion of the chest posteriorly. Roentgenograms were reported as showing a density in the right side of the chest, with the heart shifted to the right. Two days later a bronchoscopic examination was made and a considerable amount of thick purulent material was aspirated. After this procedure the temperature and the conditions in the chest became normal in three days.

CASE 15.—R. J., a 7 year old boy, had a routine tonsillectomy and adenoidectomy at Children's Memorial Hospital on June 25, 1937. Open ether anesthesia was used, and there was no preoperative medication. Three days later there began cough, fever and vomiting. On the seventh postoperative day there were a few fine rales below the spine of the right scapula and a roentgenogram showed a triangular density in the upper lobe of the right lung with slight shift of the heart (fig. 9 *A*). That same day bronchoscopy was performed by Dr. Paul Holinger, who reported that pus was found and aspirated from the bronchus of the upper lobe of the right lung. No special treatment was given, and the process resolved rather slowly (fig. 9 *B* and *C*). The condition was probably atelectasis, although it may have been a true pneumonia, when the roentgenograms and course are considered.

CASE 16.—W. B., a 7 year old boy, was admitted to the Los Angeles Children's Hospital in November 1932, with a history of pain in the abdomen for fourteen hours. He was still

Fig. 10 (case 17).—*A*, roentgenogram of the chest on the second postoperative day (the patient had exhibited symptoms of asthma before the operation); *B*, follow-up roentgenogram.

coughing badly from an infection of the upper respiratory tract, which began with high fever five days previously. Examination revealed tenderness in the right lower quadrant of the abdomen. There were some coarse rales in the bases of both lungs. Open ether anesthesia was maintained for forty-five minutes, and premedication included 1/12 grain (0.005 Gm.) of morphine sulfate and 1/250 grain (0.26 mg.) of atropine. Twenty-four hours after the operation the temperature rose suddenly to 105 F. and the patient became dyspneic and slightly cyanotic. There were dulness and tubular breathing in the bases of both lungs. He was treated with oxygen (tent) and frequent change of position. The process cleared spontaneously in three days.

CASE 17.—R. G., a 10 year old boy, was admitted to the Los Angeles Children's Hospital in March 1934, complaining of pain in the abdomen of twenty-four hours' duration. The child was asthmatic and had been coughing and wheezing for three days. Open ether anesthesia lasted for one hundred and twenty minutes. At operation a ruptured retrocecal appendix was discovered and removed. Twenty-four hours later the temperature was 104 F. and the patient was wheezing, dyspneic and slightly cyanotic. There were rales over the bases of both lungs and dulness over the left side (fig. 10 *A*). Treatment consisted of the use of an oxygen tent, administration of 1/12 grain (0.005 Gm.) of morphine sulfate for restlessness and inhalation of carbogen later. The chest and temperature were normal in seven days. A follow-up roentgenogram showed a normal chest (fig. 10 *B*).

CASE 18.—D. C., a 9½ year old girl, was admitted to the Los Angeles Children's Hospital on June 11, 1940 with a history of pain in the right lower quadrant of the abdomen for ten hours. She had vomited a number of times. Her past history included congenital syphilis and a long series of operations on a fractured right astragalus. She was operated on immediately, and a highly inflamed appendix was found. Open ether anesthesia lasted for fifty minutes. One-eighth grain (0.008 Gm.) of morphine sulfate and 1/250 grain (0.24 mg.) of atropine before the operation and morphine sulfate (⅛ grain [0.008 Gm.]) three times the next day were also given. On the second postoperative day the temperature rose to 105 F. and there were dyspnea, pain in the left side of the chest and cough. Rales were heard in the left axilla, and roentgen examination showed atelectasis on the left (fig. 11). Treatment consisted of inhalations of carbon dioxide every two hours, change of position and use of sulfapyridine. On the following day the temperature was down and the chest clearing. The child was discharged well.

CASE 19.—A. L., an 8½ year old girl, was admitted to the Los Angeles Children's Hospital on Aug. 20, 1940, with a history of pain in the abdomen and vomiting for two days. Examination revealed generalized rigidity over the abdomen and most pain over the right lower quadrant. By rectal examination a tender mass was felt on the right side. The child's mother died of tuberculosis, and the child had a positive reaction to tuberculin. At operation generalized peritonitis was found and a ruptured appendix was removed. Open ether anesthesia was main-

Fig. 11 (case 18).—Condition of the chest two days after operation. Clinically there was sudden onset of cough, pain in the left side of the chest and dulness and rales over the area. The process cleared in one day.

tained for sixty-five minutes. Her postoperative condition was fairly good, but on the second postoperative day the temperature rose to 104.8 F., there were dulness and absence of breath sounds over the right side of the chest and the heart was shifted to the right. She had six doses of morphine sulfate (1/10 grain [0.006 Gm.] each) in the thirty-six hours immediately after the operation. Inhalation of carbon dioxide and oxygen was started when the roentgenogram showed atelectasis present (fig. 12 *A*). The patient still had fever, but her chest seemed better (fig. 12 *B*) a few days later. Recovery was slow. Treatment consisted of inhalations of carbon dioxide, change of position and use of sulfapyridine. Since this time active pulmonary tuberculosis has developed, and it is probable that the atelectasis was made easier by pathologic conditions already existent in the child's chest.

CASE 20.—A. T., a 3 year old boy, was admitted to the Los Angeles Children's Hospital on Aug. 14, 1937, for an elective herniorrhaphy. His past history included eczema in infancy and asthma for one year. A physical examination just before the operation revealed normal conditions, but he had been coughing for three days. Leukocytes in the blood numbered 12,800, with a differential count including 23 per cent eosinophils. He had open ether anesthesia for forty minutes. Premedication was 1/350 grain (0.17 mg.) of atropine. Twenty-four hours after the operation there were sudden dyspnea and high fever and examination elicited dulness and diminished, high-pitched breath sounds over the right lower portion of the chest. A roent-

genogram was taken on the fifth postoperative day (fig. 13 *A*); it showed particularly atelectasis of the upper lobe of the right lung. No special treatment was given, and there was spontaneous clearing in ten days (fig. 13 *B*).

CASE 21.—F. R., an 8 year old boy, was admitted to the Los Angeles Children's Hospital in September 1937, complaining of slight pain in the abdomen for eight hours. There was no

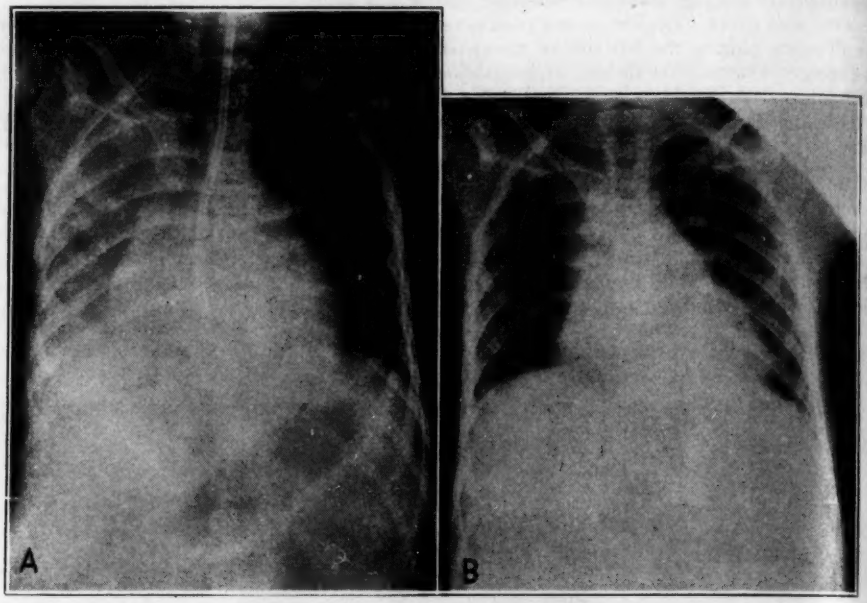


Fig. 12 (case 19).—*A*, collapse of the entire right lung forty-eight hours after operation; *B*, four days later the mediastinum has returned to the midline but the chest is not entirely normal. The girl was eventually found to have active tuberculosis.

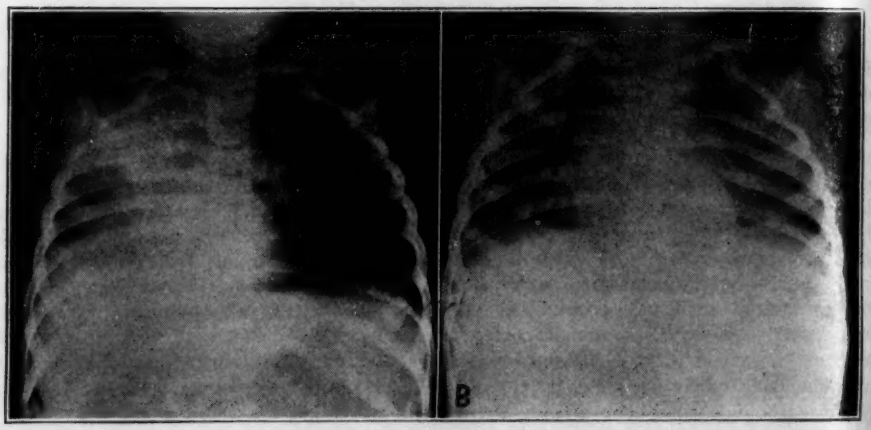


Fig. 13 (case 20).—*A*, pronounced atelectasis of the upper and lower lobes of the right lung on the fifth postoperative day; *B*, complete disappearance of atelectasis eleven days later. The patient was severely asthmatic.

vomiting. His past history included many similar attacks, often associated with attacks of asthma. On physical examination there was questionable tenderness on the right side. Leukocytes numbered 10,300, with a differential count including 14 per cent eosinophils. Because of

the past history of frequent attacks of abdominal pain, it was decided to remove the appendix, which, incidentally, was normal. Premedication was 1/10 grain (0.006 Gm.) of morphine sulfate and 1/250 grain (0.26 mg.) of atropine, and open ether anesthesia lasted one hour. Three days after the operation the child became dyspneic, the temperature rose to 103 F. and there was slight cyanosis. Examination showed the heart to be pulled to the right side, and there were dulness and suppression of breath sounds over the base of the right lung. No treatment except use of a sedative cough mixture was instituted. The process cleared spontaneously in five days.

## COMMENT

There is probably no single cause of collapse following surgical measures. Out of the maze of literature certain fairly definite facts have come to stand out over others. These will be briefly taken up.

*A. Bronchial Obstruction by a Plug of Mucus.*—In 1929, Coryllos and Birnbaum [12] established beyond doubt that in the experimental animal atelectasis develops behind bronchial obstruction provided the lung circulation remains intact. Lee and his associates [4] dramatically showed that human mucus from patients with postoperative collapse caused collapse in dogs when it was placed in their bronchi via a bronchoscope and the cough reflex was controlled. Further, there have been numerous reports in the literature, by Jackson,[13] Holinger,[14] Brown,[15] Tucker [16] and others, describing the removal by direct vision of plugs of inspissated mucus, with almost immediate areation of the involved lobe. Others have reported cases in which collapse was cured by a sharp slap on the back over the involved lobe.[9]

It is a common observation that all postoperative pulmonary complications begin with bronchitis. The mucus stagnates owing to position or to the inability of the patient to get rid of it and may inspissate and completely block a bronchus. Behind this blockade the residual air is absorbed by the blood stream. The lung now decreases in size; the normal neighboring lung comes over, and the diaphragm comes up to fill in the space left by the collapsed lung. If there is enough shrunken lung, the mediastinum and heart must go that way to equalize tension between the two sides of the chest.

Coryllos and Birnbaum expressed the opinion that injury to cilia has much to do with postoperative collapse.[12] If the cilia are functioning properly, they can do much to keep mucus moving outward to a place where it can be coughed up or swallowed. They suggested frequent colds and allergy as being factors which might decrease efficiency of the cilia in removing thick, tenacious mucus.

*B. Hypoventilation.*—Churchill and McNeil in 1927 brought attention to a great decrease in vital capacity following abdominal operations, often as much as 70 to 80 per cent of normal. Overholt [17] confirmed this and showed roentgen evidence that the diaphragm was high and its movement greatly reduced after surgical intervention. This total reduction of respiration is based partly on the pain of deep breathing, partly on the natural splinting of injured tissues and partly

13. Jackson, C., and Jackson, C. L.: Postoperative Pulmonary Complications, Internat. Clin. **4**:151-155 (Dec.) 1932.

14. Holinger, P. H.: Bronchoscopy in Postoperative Pulmonary Complications, S. Clin. North America **18**:237-248 (Feb.) 1938.

15. Brown, A. L.: Postoperative Pulmonary Atelectasis: Observations on Importance of Different Types of Bronchial Secretion and Anesthesia, Arch. Surg. **22**:976-982 (June) 1931.

16. Tucker, G.: Bronchoscopic Observations on Obstructive Pulmonary Atelectasis, Arch. Otolaryng. **13**:315-325 (March) 1931.

17. Overholt, R. H.: Postoperative Pulmonary Hypoventilation, J. A. M. A. **95**:1484-1488 (Nov. 15) 1930.

on the anesthesia itself. Recent experimental work by Galbraith and Steinberg[18] indicated that in dogs atelectasis can occur with or without bronchial obstruction if respiration is seriously restricted. If the lungs are not used, air will be absorbed and shrinkage of the lungs will occur.

*C. Decrease in Body Tonus.*—Yandell Henderson[19] and his co-workers have stressed the loss of body tone following anesthesia and surgical measures. The thoracic muscles and the diaphragm control the size of the thorax and consequent size of the lungs. If these muscles have decreased tone following a surgical operation (and he has shown by an ingenious device that they do), then the lung is relatively deflated and minute areas of collapse occur even though only a small amount of mucus may be present. Too, after an abdominal operation, some air is present in the abdomen, and this further pushes the diaphragm upward and relaxes its tone.

*D. Allergy.*—Wilmer, Cobe and Lee[20] reported 10 consecutive cases of postoperative collapse from their clinic and found that in all 10 the patients were subject to allergy (some of the cases were not too conclusive, but most were definite and included data on cutaneous tests, etc.). In the present series there were 5 cases of definite and 2 cases of questionable allergy. The proper incidence is hard to determine, however, because it must be admitted that histories taken in a surgical service are seldom completely thorough as to history of allergy.

Allergy may well be an etiologic factor in collapse, because the usual allergic response is the production of a thick, tenacious sputum of a particular kind which in physical properties is practically identical with the sputum described as present in all cases of postoperative collapse. One may postulate that the anesthetic or the surgical procedure sets off the allergic bronchial response with its attendant edema and infiltration of the bronchial walls and the production of the typical obstructing sputum. One other factor may be of importance: Coryllos stated the belief that the cilia play an important part in the production of stagnation of mucus, and he suggested allergy as an etiologic factor because in allergy, with its chronic bronchitis, the cilia are definitely damaged. Andrews[21] has shown bronchoscopically that the cilia are functionally below par in asthmatic subjects.

*E. New Closed System of Anesthesia.*—Jones and Burford[22] have recently called attention to a new factor in postoperative collapse based on the newer methods of closed anesthesia. With this method the anesthetic and oxygen are the only gases entering the system. During the course of an hour or so spilling, leaking and treatment of apnea have caused the anesthetist to add more of the anesthetic gases but not more of the inert nitrogen, which was originally present in the lungs. After a time, then, the nitrogen content is down to a negligible percentage.

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18. Galbraith, E. G., and Steinberg, B.: Developmental Mechanism of Pulmonary Atelectasis, Ann Otol., Rhin. & Laryng. **46**:800-817 (Sept.) 1937.

19. Henderson, Y.: Wesley M. Carpenter Lecture: Atelectasis, Massive Collapse and Related Post-Operative Conditions, Bull. New York Acad. Med. **11**:639-656 (Nov.) 1935.

20. Wilmer, H. B.; Cobe, H. M., and Lee, W. E.: Incidence of Allergy and Asthma in Group Developing Post-Operative Atelectasis, Ann. Surg. **91**:651-658 (May) 1930.

21. Andrews, A. H., Jr.: Bronchoscopy in Bronchial Asthma, Illinois M. J. **73**:25-31 (Jan.) 1938.

22. Jones, O. R., and Burford, G. E.: Massive Atelectasis Following Cyclopropane Anesthesia: Report of Cases and Therapy of Cause and Prevention, J. A. M. A. **110**:1092-1095 (April 2) 1938.

It has been shown recently by Baruch and others (cited in Jones and Burford's article [22]) that all the usual anesthetic gases and oxygen and carbon dioxide are absorbed rapidly from the lungs, in from one to ten minutes. On the other hand, the inert gases nitrogen, hydrogen and helium take from twenty to thirty hours for complete absorption.[23] From these figures it is easy to see what may happen at the end of a closed type of anesthesia; the only gases present in the lungs are those which are rapidly absorbed, and if they are absorbed before atmospheric nitrogen gets in to keep the bronchioles and alveoli distended collapse may occur. To make matters worse, the anesthetist produces deep respirations with carbon dioxide in oxygen, both of which are again rapidly absorbed. This is exactly what has happened recently. Burford reported 2 cases of his own and 2 others in which massive collapse occurred on the table or immediately afterward and at autopsy was shown to be the cause of death.

What probably happens is that a small amount of mucus may be enough to occlude the bronchus; the air distal to the occlusion is absorbed, and atelectasis follows. Burford makes two important suggestions: The first is to use helium (a light gas) in the anesthetic mixture to eliminate the danger of rapid complete absorption of gases; the second is to use carbon dioxide in air instead of in oxygen to wash out the lungs; the reasons are self evident from the preceding discussion.

## CONCLUSION

Summing up, postoperative collapse may be due to many conditions, or combination of conditions. Following surgical operation and anesthesia there is decreased ventilation of the lungs, a high diaphragm and a deflation of the lungs. The tonus of the whole body and especially of the muscles of respiration is seriously decreased. The patient will not breathe deeply because of the pain and because he has probably been given large doses of a sedative. Next comes the production of some mucus due to the irritation of the anesthetic gases, to allergy, to an infection of the upper respiratory tract or to causes still unknown; this mucus usually gravitates to the bronchi of the lower lobe, is not coughed up and plugging occurs. Air is absorbed distal to the mucus, and atelectasis results. When the factor of the rapidly absorbed gases is added, then a small obstruction indeed may cause the block.

The importance of atelectasis has been subject to much debate. There is excellent evidence that any type of atelectasis, no matter what its cause, may produce bronchiectasis.[24] Anspach [25] has reported cases of proved bronchiectasis following atelectasis in infancy. It is common to find bronchiectasis developing rapidly after a foreign body has produced obstruction and atelectasis of the involved lobe. Andrus [26] in a most complete monograph on the physical factors involved in bronchiectasis concluded that atelectasis is always the forerunner of bronchiectasis. In a recent paper Brennemann [24c] concurred in this view.

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23. Coryllos, P. N., and Birnbaum, G. L.: Studies in Pulmonary Gas Absorption in Bronchial Obstruction: Theory of Air Absorption in Atelectasis, Am. J. M. Sc. **183**:347-359 (March) 1932.

24. (*a*) Boyd, G. L.: Lobar Collapse in Children, J. A. M. A. **105**:1832-1835 (Dec. 7) 1935. (b) Lander, F. P. L., and Davidson, M.: Aetiology of Bronchiectasis (with Special Reference to Pulmonary Atelectasis), Brit. J. Radiol. **11**:65-89 (Feb.) 1938. (*c*) Brennemann, J.: Bronchiectasis as a Pediatrician Sees It, Dis. of Chest **19**:39-49 (Jan.-Feb.) 1943.

25. Anspach, W. E.: Atelectasis and Bronchiectasis in Children: Study of Fifty Cases Presenting Triangular Shadow at Base of Lung, Am. J. Dis. Child. **47**:1011-1050 (May) 1934.

26. Andrus, P. M.: Bronchiectasis: Analysis of Its Causes, Am. Rev. Tuberc. **36**:46-81 (July) 1937.

It is true that bronchiectasis will develop in few cases of postoperative collapse if proper treatment is employed, yet if even 1 patient can be spared this foul disease all the careful handling of the occasional patient with atelectasis will be well worth while. It is a good general rule to consider that a pulmonary complication of a surgical operation may begin to produce permanent changes in the lung if it persists unresolved for ten to fourteen days after symptoms occur. Bronchoscopy then is a necessity and should be utilized without delay.

*Prophylaxis.*—1. Before Surgical Intervention: Certainly an elective operation should be delayed until all infection of the upper respiratory tract has abated. This is logical, as production of mucus and swelling of the mucous membranes go with infections of the upper respiratory tract. Use of atropine is questionable; enough will no doubt keep down secretions during the operation, but too much may inspissate them later. A small preoperative dose of morphine is probably safe. Certainly one should be on the lookout for asthmatic children.

2. During the Anesthetic and Operation: Extremely deep anesthesia produces hypotonus and therefore must be avoided. If possible, helium may be used when the closed system of anesthesia is to be the method used. At the end of the operation gases should be washed out with carbon dioxide in air. Many surgeons use a midline incision and parallel incisions instead of a paramedian one, as there is less pain on breathing. Cutting the oblique muscles is bad, because these are accessory muscles of respiration (Brock).

3. Postoperative: Atropine should never be used. The use of morphine is debatable; there is no question but that the drug depresses the cough reflex, but, on the other hand, it allows the patient to get some relief from pain and therefore to cough out his secretions. Probably the best answer is to use it moderately but certainly not as a *pro re nata* order. The patient's position should be changed frequently. It has been shown that a patient may breathe easier when propped up, but it is harder to cough in this position. The child should be put on his side without pillows and urged to cough; the nurse may exert pressure on the dressings of the wound to ease the pain of coughing.

*Treatment After Collapse Occurs.*—The main procedure in the treatment of postoperative collapse is to help the patient cough out the secretions. This is best done by frequent changes of position, but with the involved side uppermost most of the time. Potassium iodide or ammonium chloride may be given to liquefy secretions. For allergic patients epinephrine hydrochloride may help to open the bronchi and reduce swelling.

Henderson's work on tonus of muscles included experiments with strychnine following surgical operation. He found that the tonus of muscles was brought up to normal and above by $\frac{1}{400}$ grain (0.15 mg.) in adults. (I have no personal experience with this type of treatment, but it is worth a trial.)

Inhalations of carbon dioxide as treatment and prophylaxis are questioned both clinically and theoretically. King in a study of a large series of cases at Massachusetts General Hospital showed that alternate patients did no better than the controls, who were not given carbon dioxide. Broock cited studies theorizing that inhalations of carbon dioxide may tend to suck or push secretions farther down the bronchi. It seems reasonable that inhalations of carbon dioxide would overcome the element of hypoventilation at least and might set up a productive cough.

If none of these measures are effective within a few days, then bronchoscopic drainage should be instituted without delay.

The following tabulation shows precautions against and treatment for postoperative collapse based on probable etiologic factors:

| *Probable Causes of Postoperative Atelectasis* | *Precautions and Treatment* |
|---|---|
| 1. Premedication | Give short-acting barbiturates; use little if any atropine. |
| 2. Infection of the upper respiratory tract or chronic bronchitis | Delay the operation if possible; take special precautions to promote drainage after the operation; administer potassium iodide postoperatively |
| 3. Allergic reaction | Take adequate history; take special precautions to promote drainage after the operation; possibly use epinephrine hydrochloride |
| 4. Length and depth of anesthesia | Elicit the surgeon's cooperation; keep the anesthesia as light as possible |
| 5. Type of operation | Use a transverse incision if an operation on the upper part of the abdomen is to be performed |
| 6. Hypotonus of respiratory muscles | Use short anesthesia and operation and as little postoperative medication as possible; give frequent help in turning and breathing; use of strychnine is questionable |
| 7. Hypoventilation | Stop the anesthetic before the operation is finished; wash out the lungs with carbon dioxide in air and not carbon in oxygen; urge the child to cough frequently; change his position frequently; if collapse or bronchial moisture is developing use the Trendelenburg position, administer carbon dioxide and urge the child to cough and turn |
| 8. Absorption of gases when closed system of anesthesia is used | Wash out the lungs with carbon dioxide in air, not in oxygen |
| 9. Mucus plug | Suck out all possible secretions at the end of the operation; urge the patient to cough by turning and use of carbon dioxide; reduce the pain by using small doses of opiates; if collapse occurs, put the involved side uppermost and use bronchoscopy without delay if the atelectasis persists |

## SUMMARY

Twenty-one cases of postoperative atelectasis in children have been presented; the cases were taken from the records of two large children's hospitals over a ten year period.

Etiologic factors for collapse following surgical procedures are considered, and a plan of treatment based on these several factors is offered.

The importance of atelectasis in the production of bronchiectasis is stressed.

Bronchoscopic drainage in cases of persistent collapse is emphasized as the proper treatment.

Dr. Joseph Brennemann has served as critic and guide in this study. He personally cared for most of the patients whose cases are included in this report, and many of his ideas on the cause and treatment of the condition form the basis for this paper.

Lieut. Lloyd Harris (MC), U.S.N., formerly head resident at the Children's Memorial Hospital, prepared the statistical and roentgen data.

6507 Lindenhurst, Los Angeles.

# Abstracts from Current Literature

## Anatomy and Physiology

EFFECT OF ANOXIA ON BLOOD SUGAR IN ADRENALECTOMIZED-PANCREATECTOMIZED DOGS. IRVINE MCQUARRIE, M. R. ZIEGLER and L. J. HAY, Proc. Soc. Exper. Biol. & Med. **49**:291 (Feb.) 1942.

The investigation was undertaken to determine whether or not a previously observed fall in blood sugar in adrenalectomized animals resulting from anemia was due to stimulation of the vagoinsulin system. Two adrenalectomized-pancreatectomized dogs were repeatedly made to breathe an atmosphere composed of 5 per cent oxygen and 95 per cent nitrogen for periods of two and a half hours. Blood sugar values were determined at thirty minute intervals. These showed no significant fall as compared with values obtained for the same animals kept in room air. The results were interpreted as evidence that the hypoglycemic effect of anoxia in adrenalectomized dogs is due to vagoinsulin stimulation.

HANSEN, Minneapolis.

PHYSIOLOGICAL EFFECTS OF BLAST. P. L. KROHN, D. WHITTERIDGE and S. ZUCKERMAN, Lancet **1**:252 (Feb. 28) 1942.

A series of experiments in which rabbits' trunks were protected while their heads were directly exposed to high blast pressures confirmed earlier observations that thoracic and abdominal lesions resulting from blast are due to the wave acting on the surface of the body and not to either the pressure or the suction components of the wave acting through the nose and mouth. The thoracic and abdominal lesions are primarily due to the impact of the pressure component on the body wall, and the lesions can occur when the suction component is excluded.

Animals exposed to blast show an immediate fall in arterial blood pressure. There is usually a prolonged and great increase in the respiratory rate of rabbits which survive exposure to blast.

The clinical implications discussed include the importance of an accurate history of the circumstances of the injury and the state of the ear drum and middle ear when signs of pulmonary and abdominal damage exist. Rapid shallow breathing or slow labored breathing may be expected.

LANGMANN, New York.

## Biochemistry; Bacteriology, and Pathology

THE EFFECT OF VARIOUS SULFONAMIDES ON HEMOLYTIC STAPHYLOCOCCUS MEASURED WITH THE MANOMETRIC TECHNIQUE. WALTER KEMPNER, C. SCHLAYER and PERCY SUMMERS, Am. J. M. Sc. **203**:172 (Feb.) 1942.

The effect of various sulfonamides on the growth rate of three pathogenic strains of hemolytic staphylococci was determined by measuring manometrically the oxygen consumption and anaerobic formation of carbon dioxide of dextrose-peptone-broth cultures. The order of potency of the various drugs tested was as follows: least effective, sulfaguanidine, with a growth of bacteria after four and one-half hours which corresponded to an oxygen consumption of 665 cubic millimeters per hour, as compared with 865 cubic millimeters of the drug-free control; then sulfapyridine sodium, with 623 cubic millimeters; sodium disulon (sodium N4-sulfanilylsulfanilamide), with 534 cubic millimeters; sulfadiazine, with 402 cubic millimeters, and sulfathiazole and sulfathiazole sodium, with 203 and 182 cubic millimeters respectively.

The amount of staphylococcic growth in sulfapyridine sodium was more than 200 per cent in excess of that in sulfathiazole, while that in sulfadiazine was only 100 per cent. In cultures of pneumococci examined under the same experimental conditions sulfapyridine sodium was definitely superior to sulfadiazine. For both bacteria sulfathiazole was the most potent inhibitor.

By varying the concentrations of the drugs it could be shown that sulfadiazine was only half as effective as sulfathiazole as an inhibitor of the growth of staphylococci. The sulfonamide compounds inhibit the growth rate of staphylococci not only at optimal but at lowered oxygen concentrations and under anaerobic conditions.

HENSKE, Omaha.

THE KILLING OF BACTERIAL SPORES IN FLUIDS BY AGITATION WITH SMALL INERT PARTICLES. HAROLD R. CURRAN and FRED R. EVANS, J. Bact. **43**:125 (Feb.) 1942.

Meltzer was the first to suggest that certain vegetative bacteria contained in broth or salt solution could be killed when subjected to long and vigorous mechanical shaking in contact with finely divided materials. This was back in 1892, and since then little attention has been given to mechanical agencies capable of killing bacteria. However, the authors have attempted to emphasize again the germicidal efficiency of mechanical methods. Their experiments have shown that bacterial spores and vegetative forms are progressively destroyed when the fluid suspensions are agitated long and vigorously with finely divided abrasives. Spores so treated die at a rate which corresponds with the unimolecular law for chemical reactions. The cumulative result of abrasion and repeated collisions between the abrasive particles and the bacteria is a gradual disintegration of the cells. This is attended by a material decrease in opacity of the suspension, a fact which indicates that the cells are reduced to a very fine state of division. Highest sporicidal efficiency was obtained with small glass beads. These observations should be of definite value in the preparation of vaccines.

STOESSER, Minneapolis.

THE ISOLATION FROM SOIL OF SPORE-FORMING BACTERIA WHICH PRODUCE BACTERICIDAL SUBSTANCES. J. L. STOKES and C. R. WOODWARD JR., J. Bact. **43**:253 (Feb.) 1942.

The isolation of crystalline bactericidal substances from a soil bacillus has aroused considerable interest. The isolated material was found to be bactericidal in extremely small amounts. Gram-positive bacteria were affected; the gram-negative organisms were not harmed. One of the first substances was called gramicidin, and later two more were found, namely tyrothricin and tyrocidine hydrochloride. They exhibited a high degree of bactericidal activity in vitro against both gram-negative and gram-positive bacteria. In all these investigations the bacilli capable of destroying other bacteria were obtained either accidentally or by indiscriminately isolating strains and testing each for antibacterial properties. In the authors' present report a simple rapid method for isolating from soil bacteria which produce bactericidal agents is described. The characteristics of these bacteria and the bacteriostatic and the bactericidal properties of the agents obtained from them are also presented. For full details the original article should be consulted.

STOESSER, Minneapolis.

INVESTIGATION ON THE IMMUNOGENIC PROPERTIES OF FIXED RABIES VIRUS STRAINS. BETTYLEE HAMPIL and G. H. ROBERTS, J. Bact. **43**:397 (March) 1942.

The authors investigated 17 strains of fixed rabies virus with respect to their immunogenic properties as virulent vaccines in 21 day old Swiss mice. Immunity was tested by intracerebral injection of homologous virus. Their results indicated that wide differences occur in the immunity response of the mice depending on the strain of virus employed. The practical application of these studies may be significant in the preparation of rabies vaccines. The use of mice in the weeding out of nonantigenic vaccines is now an established procedure. A vaccine prepared from a strain possessing high intrinsic immunogenic properties as a living virus may show superior antigenic potency to a vaccine prepared from a strain less immunogenic as a living virus when the homologous strain is used as the challenge virus. Killed vaccines prepared from a highly immunogenic strain will produce on repeated administration a greater degree of immunity to heterologous strains than those prepared from strains possessing low initial immunogenic properties. Studies along these lines are being continued.

STOESSER, Minneapolis.

INFLUENCES OF ERYTHROCYTES AND OF LEUKOCYTES ON STABILITY AND TRANSFER OF ASCORBIC ACID IN HUMAN BLOOD. MARTIN HEINEMANN, J. Clin. Investigation **20**:467 (Sept.) 1941.

The deterioration of ascorbic acid in serum in an atmosphere of air was prevented by the presence of intact erythrocytes and enhanced by the presence of leukocytes. Under nitrogen, ascorbic acid added to serum was transferred to leukocytes but not to erythrocytes. Erythrocytes did not take up ascorbic acid in an atmosphere of air. Data on the concentrations of ascorbic acid in different layers from the same serum, obtained by ultracentrifuging, were presented to show that ascorbic acid is not bound to serum proteins and that different concentrations of protein do not affect the rate of deterioration of serum ascorbic acid.

GUEST, Cincinnati.

STUDIES ON SULFONAMIDE-RESISTANT ORGANISMS: I. DEVELOPMENT OF SULFAPYRIDINE RESISTANCE BY PNEUMOCOCCI. L. H. SCHMIDT, CLARA SESLER and H. A. DETTWILER, J. Pharmacol. & Exper. Therap. **74**:175 (Feb.) 1942.

Certain strains of pneumococci were made highly resistant to sulfapyridine by passage through series of white mice. A similar resistant strain was obtained by growing the strain in culture mediums containing the drug, and this strain was resistant in vivo. Resistance to sulfapyridine was retained by these strains for more than two hundred passages through untreated mice.

PILCHER, Cleveland.

## Metabolism; Infant Feeding; Milk and Other Foods

DIETARY PROTEIN AND THE REGENERATION OF SERUM ALBUMIN: IV. THE POTENCY VALUES OF DRIED BEEF SERUM, WHOLE EGG, COW'S MILK, COW'S COLOSTRUM, LACTALBUMIN, AND WHEAT GLUTEN. A. A. WEECH, Bull. Johns Hopkins Hosp. **70**:157 (Feb.) 1942.

With his method of assaying food proteins for efficiency in promoting the formation of serum albumin the author found that drief beef serum and lactalbumin have potency values of 0.739 and 0.773 and could be called superior protein for synthesis of serum albumin. Whole egg (0.602) and cow's colostrum (0.611) are good proteins; cow's milk (0.483) is fairly well utilized, while wheat gluten (0.207) is a poor protein for synthesis of serum albumin.

LYTTLE, New York.

THE IRON REQUIREMENT OF CHILDREN OF THE EARLY SCHOOL AGE. FRANCES A. JOHNSTON and LYDIA J. ROBERTS, J. Nutrition **23**:181 (Feb.) 1942.

Johnston and Roberts concluded from their studies with 21 children 8 to 11 years of age that good hemoglobin levels could be maintained with an iron intake of 11.4 mg. per day, or 0.35 mg. per kilogram of body weight, if the rest of the diet was adequate.

FREDEEN, Kansas City, Mo.

THE IRON METABOLISM AND REQUIREMENT OF YOUNG WOMEN. RUTH M. LEVERTON and ALICE G. MARSH, J. Nutrition **23**:229 (March) 1942.

Leverton and Marsh made ninety-nine one-week studies on the iron metabolism of 69 women (16 to 27 years of age) who were living on self-chosen diets. The daily iron intake of 10.44 mg. provided an average daily storage of 1.37 mg., which was ample to replace normal menstrual losses. These investigators suggest that emphasis should be placed on a well balanced optimum diet for such women rather than on an adequate amount of iron in the diet, since the former has been shown to function in efficient iron absorption and utilization.

FREDEEN, Kansas City, Mo.

QUANTITATIVE DISTRIBUTION OF PHOSPHORUS AND CALCIUM IN CERTAIN FRUITS AND VEGETABLES. H. C. SHERMAN and M. S. RAGAN, J. Nutrition **23**:283 (March) 1942.

All of the foods in this series were the varieties which may be eaten with or without their seeds. Quantitative studies were made by Sherman and Ragan for the relative amounts of phosphorus in the flesh and seeds of seven species of fruits; in the pods and ovules of four varieties of beans, and in the leaves, edible stems and flowerbuds of broccoli; they determined the relative amounts of calcium in the same parts of broccoli and in the septums and flesh (pulp and juice) of oranges. Four tables are included showing the results.

FREDEEN, Kansas City, Mo.

EFFECT ON GROWTH AND CALCIUM ASSIMILATION OF CITRIC ACID–POTASSIUM CITRATE MIXTURES. CAROLINE SHERMAN LANFORD, J. Nutrition **23**:293 (March) 1942.

Lanford concluded that orange juice favors calcium assimilation in the growth process of young rats through some factor other than its content of citric acid and citrates and preponderance of base-forming elements.

FREDEEN, Kansas City, Mo.

DEMONSTRATION OF RH ANTIBODY IN BREAST MILK. ERNEST WITEBSKY, GEORGE W. ANDERSON and ANNE HEIDE, Proc. Soc. Exper. Biol. & Med. **49**:179 (Feb.) 1942.

Breast milk from the mother of an infant having progressing jaundice and anemia, with 58 nucleated red blood cells per hundred white blood cells, was found to have Rh anti-

bodies, but the titer was low in comparison with that of the mother's serum. The infant improved after two transfusions and the discontinuance of breast feeding.

HANSEN, Minneapolis.

MILK A HUMAN POISON. M. T. DAVIDSON, South. M. J. **35**:196 (Feb.) 1942.

The author reported 120 cases of allergic reactions in which the major role of production of symptoms was assigned to less than a dozen allergens. Many other allergens were almost as frequent reactors. For instance, the condiments chocolate, pepper, mustard and others are frequent reactors but are rarely proved to be major factors in producing symptoms.

There were four foods which were frequently blamed for producing allergic symptoms. In the order of their frequencies, for subjects above the age of infancy, they were: milk, 40 per cent; egg, 27 per cent; wheat (grains), 8 per cent, and cottonseed, 6 per cent. Somewhat larger percentages of reactions were chargeable to the same foods in the younger group.

In any allergic syndrome of perennial occurrence there is a 40 per cent or better chance that milk plays a leading role in the production of symptoms.

SCHLUTZ, Chicago.

EDEMA SYNDROME IN INFANCY. J. SCHWARZENBERG L. and S. COSTA, Rev. chilena de pediat. **12**:341 (May) 1941.

The authors report 13 cases of the unusual condition known as the edema syndrome. It seems to be particularly prevalent in Chile. In all of the cases there were edema, hypoproteinemia, hypocholesteremia, severe anemia and great feebleness. Some of the patients showed pellagroid symptoms and pellagroid prescorbutic symptoms, but this was not a consistent finding in all cases.

The authors were under the impression that in most of the cases there was some vitamin A deficiency. Renal involvement was found in only 1 of the cases and was of an infectious and septic type.

SCHLUTZ, Chicago.

## Vitamins; Avitaminoses

INTERRELATED VITAMIN REQUIREMENTS: KIDNEY DAMAGE, ADRENAL HEMORRHAGE AND CARDIAC FAILURE CORRELATED WITH INADEQUACY OF PANTOTHENIC ACID. G. C. SUPPLEE, R. C. BENDER and O. J. KAHLENBERG, Endocrinology **30**:355 (March) 1942.

In rats dietary components, vitamin intake, state of development and maturity determined the quantitative need for pantothenic acid.

Pantothenic acid appears to be required for maintaining the integrity of the adrenal cortex. Its absence or inadequacy in experimental diets results in a characteristic pathologic change of the cortical layer readily detectable by macroscopic and histologic examination.

JACOBSEN, Buffalo.

EFFECT OF CONTROLLED ASCORBIC ACID INGESTION UPON URINARY EXCRETION AND PLASMA CONCENTRATION OF ASCORBIC ACID IN NORMAL ADULTS. CLARA A. STORVICK and HAZEL M. HAUCK, J. Nutrition **23**:111 (Feb.) 1942.

A definite correlation between the daily urinary excretion of ascorbic acid and the concentration of ascorbic acid in the plasma with intakes of 75 mg. and less was noted by Storvick and Hauck, but no consistently significant correlation resulted with higher levels of ascorbic acid intake. Six normal healthy adults were the subjects used in these experiments. Supplements of 65 to 150 mg. of ascorbic acid were required by the subjects, in addition to 10 mg. in the basal diet, to maintain tissue saturation.

FREDEEN, Kansas City, Mo.

THE INACTIVATION OF VITAMIN $B_1$ IN DIETS CONTAINING WHOLE FISH. R. G. GREEN, W. E. CARLSON and C. A. EVANS, J. Nutrition **23**:165 (Feb.) 1942.

Green, Carlson and Evans after experimentation with red fox pups conclude that the actual amount of thiamine that a pound of carp inactivates is not a constant value but depends on the amount of thiamine mixed with the fish. This inactivation suggested adsorption rather than simple chemical reaction between the two substances. Foxes fed 20 per cent whole carp and given 10 mg. of thiamine hydrochloride daily did not acquire "Chastek paralysis," and the paralysis manifested itself only as a transient anorexia among those given 5 mg. daily.

Foxes given fish muscles, as carp fillet, showed no evidence of "Chastek paralysis," but those fed the fins, scales, skin and skeleton acquired "Chastek paralysis," as did the group fed the carp viscera.

FREDEEN, Kansas City, Mo.

THE ASCORBIC ACID REQUIREMENTS OF CHILDREN. OTTO A. BESSEY and RUTH L. WHITE, J. Nutrition **23**:195 (Feb.) 1942.

In this investigation, Bessey and White determined the blood plasma ascorbic levels of 93 healthy children (5 to 13 years of age) who were receiving variable amounts of ascorbic acid in their food. Three ounces (90 cc.) of orange juice or its equivalent (40 to 50 mg. ascorbic acid) plus the small amount of ascorbic acid obtained in other foods (approximately 10 mg. per day) was sufficient to produce an optimal postabsorptive plasma ascorbic acid concentration for the average child of this age group.

FREDEEN, Kansas City, Mo.

ANTIRACHITIC PROPERTIES OF A. T. 10 FOR THE RAT AND CHICKEN. JOHN T. CORRELL and E. C. WISE, J. Nutrition **23**:217 (March) 1942.

Correll and Wise concluded that the efficiency of dihydrotachysterol as to its antirachitic effects in chicks when compared on a rat unit basis was equal to that of the vitamin D from cod liver oil. The action of dihydrotachysterol resembled that of vitamin D rather than that of a parathyroid extract.

FREDEEN, Kansas City, Mo.

SERUM PHOSPHATASE IN EXPERIMENTAL SCURVY. HARRY SHWACHMAN and BERNARD S. GOULD, J. Nutrition **23**:271 (March) 1942.

Shwachman and Gould noted an immediate and continued decline in "alkaline" serum phosphatase activity reaching extremely low levels when a scorbutogenic diet was fed guinea pigs. However, rabbits able to synthesize ascorbic acid did not show the decline when fed this diet. Ascorbic acid supplementation (0.25 to 12.0 mg. daily) produced rapid increases in serum "alkaline" phosphatase activity. "Acid" phosphatase or inorganic phosphate of the serum of scorbutic guinea pigs was apparently not altered.

FREDEEN, Kansas City, Mo.

THE RATE OF INCREASE OF BLOOD PLASMA ASCORBIC ACID AFTER INGESTION OF ASCORBIC ACID (VITAMIN C). E. NEIGE TODHUNTER, RUTH C. ROBBINS and JENNIE A. MCINTOSH, J. Nutrition **23**:309 (March) 1942.

Five adults were studied by Todhunter, Robbins and McIntosh for rate of increase in plasma ascorbic acid after ingestion of 50 mg. of this vitamin. Close similarity in response was noted in all the studies. The plasma curve began to rise within thirty to sixty minutes after ingestion of 50 mg. of the crystalline ascorbic acid or its equivalent in fruit and returned to the fasting level in three to four hours. The maximum level was reached within ninety minutes after ingestion of the crystalline vitamin or orange juice. The ingestion of fat or of iron salts at the same time as the ascorbic acid did not affect the curve for increase of plasma ascorbic acid.

FREDEEN, Kansas City, Mo.

ASCORBIC ACID REQUIREMENT OF INDIVIDUALS IN A LARGE INSTITUTION. M. K. HORWITT, Proc. Soc. Exper. Biol. & Med. **49**:248 (Feb.) 1942.

Study of over 1,000 samples of blood from patients in a mental disease hospital showed that the ascorbic acid level averaged 0.40 mg. per hundred cubic centimeters, with the lowest value, 0.20 mg., obtained in April, May and June and the highest, 0.59 mg., in October and November. In a study of 40 young adults with schizophrenia, of whom 20 were given extra ascorbic acid and 20 served as controls, it was found that 50 mg. every forty-eight hours brought the blood ascorbic acid level to 0.7 mg. per hundred cubic centimeters. The daily diet contained 25 mg. From these data the average daily requirement of ascorbic acid seems to be 50 mg. In institutions it may be necessary to add ascorbic acid as such to supply vitamin C.

HANSEN, Minneapolis.

PYRUVIC ACID TEST FOR VITAMIN $B_1$ DEFICIENCY IN CHILDREN. H. ELLIS C. WILSON, Lancet **1**:199 (Feb. 14) 1942.

Wilson found that the value for blood pyruvic acid of normal children of the hospital class in England corresponds to the figure found for a similar class in America, which sug-

gests that there is no deficiency of vitamin $B_1$ in the diets. For a few children with gastro-enteritis, nephritis or convulsions, high values were found, although there were no clinical signs of vitamin $B_1$ deficiency.

LANGMANN, New York.

RELATION OF ASCORBIC ACID TO THE VITAMINS A AND D. ALF ELMBY, Nord med. **10**:1519 (May 17) 1941.

Experiments on guinea pigs have shown that there is no antagonism between pure vitamin A and D on one side and vitamin C on the other side. Cod liver oil has, however, a depressing influence on the antiscorbutic effect of ascorbic acid. This influence is thought to depend on a substance in cod liver oil which is neither vitamin A nor vitamin D.

WALLGREN, Göteborg, Sweden.

VITAMIN D AND OSSIFICATION. V. VON KRAEMER and B. LANDTMAN, Nord. med. (Finska läk.-sällskhandl.) **10**:1611 (May 24) 1941.

The authors have investigated to determine whether vitamin D has any influence in vitro on the resorption of calcium and phosphorus in the bones of rats. They found that a normal bone has the faculty to absorb from an 11 per cent solution of calcium an amount of calcium that corresponds to the weight of the bone. Bones from rats with hypervitaminosis D show an increased absorption of calcium, and its fixation is more intense. After a rachitogenic diet the absorption of calcium was diminished and its fixation was decreased. The authors are of the opinion that vitamin D has a direct influence on the vital activity of the osteoblasts.

WALLGREN, Göteborg, Sweden.

## Hygiene; Growth and Nutrition; Public Health

THE PART OF THE PRIVATE PEDIATRICIAN IN THE SCHOOL HEALTH PROGRAM. WILLIAM E. AYLING, New York State J. Med. **41**:1949 (Oct. 1) 1941.

The method of making health examinations in the schools of Syracuse, N. Y., is well outlined. It is especially important that routine examination be made of vision and hearing, in order that the proper treatment may be given. Crippled children and those with cardiac diseases need to be detected and placed in the proper schools. Parents are urged to take children to their own private physician or pediatrician. The pediatrician is urged to make these examinations carefully when required to do so and is especially requested to fill out the blanks provided by the state department of education. His cooperation and support are necessary to make the school health program a success.

AIKMAN, Rochester, N. Y.

DISCUSSION ON THE EFFECTS OF WAR TIME RATIONING ON CHILD HEALTH. Proc. Roy. Soc. Med. **35**:273 (Feb.) 1942.

From a comparison of data obtained in an unpublished dietary survey carried on during the years 1935 to 1939 on 1,000 middle class children and the actual rations of 1941, Dr. E. M. Widdowson found that the rations provide children up to 8 years of age with as much meat, bacon, sugar and jam as they were having before the war. Children over 8 were formerly eating more of all these foods, and adolescent boys were eating two or three times as much as their present rations. These children have maintained their calories by an increased consumption of bread, potatoes and other plentiful foods. The loss of protein, iron and vitamin B, brought about by the rationing of meat and bacon, is readily made good by consumption of National wheatmeal or wholewheat bread. If the margarine is fortified with vitamins so that it is equal to summer butter, children up to the age of 12 years obtain more of the fat-soluble vitamins than they did before the war. Vitamin C has been much curtailed on account of the shortage of fruits, and it is not known yet whether this is a serious matter or not.

Dr. Dagmar Curjel Wilson gave the following signs of dietary deficiency under wartime conditions in English schools: (1) follicular hyperkeratosis; (2) angular stomatitis; (3) xerosis and keratomalacia; (4) bleeding gums, and (5) rickets.

Dr. Helen Mackay stated it is difficult to arrive at a general conclusion as to wartime feeding as food conditions differ widely at different periods as well as in different areas. Also she felt there is a paucity of available facts. She said that from facts gathered by pediatricians and doctors in the Public Health Service tuberculosis among children had increased during the two and a half years of war and was still increasing, and that nutritional

anemia was also on the increase. Just from impression Dr. Mackay felt there was some increase in the incidence of rickets.

Drs. W. W. Payne, J. A. Glover and A. St. G. Hugget also took part in the discussion.

WILLIAMSON, New Orleans.

## Newborn

OPHTHALMIA NEONATORUM: THE ETIOLOGY IN 62 CONSECUTIVE CASES. S. H. McKEE, Am. J. Ophth. **25**:52 (Jan.) 1942.

McKee found cytoplastic inclusion bodies in 42 of 62 cases of ophthalmia neonatorum. He concludes that inclusion blennorhea forms a large part of the ophthalmia of the newborn. He has found sulfanilamide administered by mouth beneficial, as it is also in cases of gonococcic and of mixed infection.

W. S. REESE. [ARCH. OPHTH.]

MECHANISMS IN THE MAINTENANCE OF LIFE IN THE NEWBORN DURING ANOXIA. H. E. HEIMWICH, A. O. BERNSTEIN, H. HERRLICH, A. CHESTER and J. F. FAZEKAS, Am. J. Physiol. **135**:387 (Jan.) 1942.

In a previous publication the extraordinary tolerance of the newborn of various species to anoxia was described. The factor of anaerobic sources of energy is considered in this study.

The importance of carbohydrate as an anaerobic store of energy has been studied in the newborn rat. An increase of the carbohydrate stores permits longer survival in nitrogen while a decrease has the reverse effect. The injection of iodoacetate or fluoride shortens the survival period in nitrogen, presumably by breakdown of carbohydrate. The rate of cerebral glycolysis in the newborn animal is slower than that of the adult. The slow rate appears adequate to support the lower metabolic demands of the infant brain for a considerable period in the absence of oxygen.

NOURSE, Cleveland.

NEONATAL ASPHYXIA. HOWARD J. MORRISON, J. M. A. Georgia **31**:88 (March) 1942.

This article presents a summary of the cases of neonatal asphyxia and stresses the need for conservation by obstetricians in the use of analgesics and anesthetics during labor.

The writer feels that lay articles such as appeared in the *Reader's Digest* about painless childbirth have a bad influence on a prospective mother and that this evil influence can be answered only by a frank discussion between the attending obstetrician and the mother. After all, the mother's primary concern is having a normal, strong infant, and her secondary concern is whether or not her labor is painful.

MORRISON, Savannah, Ga.

SULPHAPYRIDINE IN OPHTHALMIA NEONATORUM. ARTHUR SORSBY, ELIZABETH L. HOFFA and ELSPETH W. SMELLIE, Brit. M. J. **1**:323 (March 7) 1942.

This article is a report of the use of sulfapyridine in the treatment of ophthalmia neonatorum. There was a total of 322 cases. In 273 of these cases treatment consisted in administration of sulfapyridine by mouth. The remaining 49 cases constituted an earlier group in which treatment had been local. The incidence of gonococcic infection for the whole group was 27.3 per cent. The treatment in the cases was the same whether the patient had gonococcic infection or not. Cultures revealed that the majority of organisms other than gonococci were staphylococci. The next most common organisms were diphtheroid bacilli, and streptococci, gonococci and the usual other organisms were also found. The dose of sulfapyridine administered was an initial dose of 0.25 Gm. which was followed by 0.125 Gm. every four hours day and night. Besides the administration of sulfapyridine the usual treatment for ophthalmia neonatorum was carried out, with irrigation and the instillation of atropine. The results of this study were as follows: Of the patients treated only with local treatment 15 per cent were cured within eight days; 58 per cent, between eight and thirty days, and 26 per cent were cured only after thirty days of treatment. Of the patients treated with sulfapyridine those cured within eight days were 61 per cent; within eight to thirty days, 32 per cent and only 5.9 per cent resisted treatment for more than thirty days.

In their summary the authors state that "sulphapyridine has proved as effective in non-gonococcal ophthalmia as in the gonococcal variety."

BIRDSONG, University, Va.

THYMIC HYPERTROPHY AND THE ROENTGEN ASPECTS OF THE HEART IN THE NEWLY BORN. P. J. AMAYA, Rev. Colomb. de pediat. y puericult. **1**:138 (Feb.) 1942.

In what amounts to a textbook article Amaya ascribes to the thymus a number of physiologic and pathologic properties which it has long been known not to possess. The

clinical and roentgen features of thymic enlargement are described, and the differential diagnosis is considered. Marfan and Paltauf are cited as authors of the assertion that enlargement of the thymus may be due to syphilis and rickets. Depending on the cause, antisyphilitic measures, vitamin D and roentgen irradiation are recommended in treatment. Attacks of asphyxia in pertussis may be due to thymic enlargement.

McCUNE, New York.

## Acute Contagious Diseases

KERATITIS DUE TO MUMPS. R. W. DANIELSON and J. C. LONG, Am. J. Ophth. **24**:655 (June) 1941.

Danielson and Long draw the following conclusions:

"Descriptions of seven cases of keratitis following mumps have been found in the literature, and one new case has been observed by the writers. There is sufficient similarity in the onset, course and appearance of these cases to justify considering this condition as a definite clinical entity. The disease affects one eye only and begins from 2 to 11 days after the onset of the parotid swelling. The cornea is rather uniformly grayish-white with very minute white interlacing fibrillae. Pain is not severe, but photophobia and lacrimation are present. Vision is markedly reduced. Improvement occurs with astonishing rapidity, resulting in complete recovery within a month or less after onset. There is no vascularization nor scarring of the cornea. The uvea is apparently not involved in the inflammatory process."

W. S. REESE [ARCH. OPHTH.].

LITTLE PROGRESS IN DIPHTHERIA IMMUNIZATION. Health News **19**:52 (March 23) 1942.

A list is given of fifty-two communities in the state of New York in which 35 per cent of the children under 5 years of age have been immunized against diphtheria. Little progress has been made in the last two years in increasing the percentage of children under 5 years of age immunized against this disease. Upstate New York did, however, establish a new low record, with only 69 cases of diphtheria reported in 1941. The state health department warns that the movement of nonimmune persons seeking employment due to the current war effort may increase diphtheria and that every effort should be made to promote immunization against diphtheria and smallpox.

AIKMAN, Rochester, N. Y.

SCARLET FEVER. ARCHIBALD L. HOYNE, Illinois M. J. **81**:12 (Jan.) 1942.

Hoyne discusses the history, portals of entry, transmission, age incidence, symptoms, diagnosis, complications, mortality and treatment of scarlet fever. He believes that if tonsils and adenoids were removed before children enter school there would be less scarlet fever. It seems advisable to perform a Dick test on every child before he enters school. Human convalescent serum and scarlet fever antitoxin are of value in active treatment.

BARBOUR, Peoria, Ill.

THE DIAGNOSIS OF POLIOMYELITIS. BENJAMIN M. LEVIN, W. H. REALS, I. P. BRONSTEIN and M. MAGREE, Illinois M. J. **81**:71 (Jan.) 1942.

From Jan. 1, 1935 to Dec. 31, 1940, 292 patients were admitted to the Cook County Hospital with the diagnosis of poliomyelitis. Of the initial diagnoses, 24.4 per cent were later found to be incorrect.

During the systemic phase of poliomyelitis it is impossible to differentiate its onset from that of various other acute infections. Infections of the upper respiratory tract are the most frequent sources of error. During the summer and early fall, patients with infections of the upper respiratory tract and with gastroenteritis should be kept under observation until poliomyelitis has been ruled out.

The various other conditions confused with poliomyelitis can be diagnosed by careful history, clinical examination and examination of the spinal fluid.

BARBOUR, Peoria, Ill.

POLIOMYELITIS IN AN INFANT FOUR MONTHS OF AGE DIAGNOSED IN THE PRE-PARALYTIC STAGE. JULIUS H. HESS, SIDNEY O. LEVINSON and EDMOND R. HESS, Illinois M. J. **81**:124 (Feb.) 1942.

Hess and co-workers report a case of poliomyelitis in a 4 month old child diagnosed in the preparalytic stage.

BARBOUR, Peoria, Ill.

COMMENTS ON EARLY DIAGNOSIS AND TREATMENT OF ANTERIOR POLIOMYELITIS. ARTHUR J. FLETCHER, Illinois M. J. **81**:152 (Feb.) 1942.

Fletcher discusses the difficulties of diagnosis and the lack of indications of specific therapy in cases of early acute poliomyelitis and mentions the lack of available specific remedies.

BARBOUR, Peoria, Ill.

THE IMMUNOLOGIC RESPONSE OF ALLERGIC CHILDREN TO TOXOID. TOWNSEND B. FRIEDMAN, JOHN A. BIGLER and MARIE A. WERNER, J. Allergy **13**:114 (Jan.) 1942.

A comparison of the immunologic response of a group of allergic and nonallergic children to the combined tetanus and diphtheria alum-precipitated toxoids and to alum-precipitated tetanus toxoid was made.

The children were divided into several groups: One group received two 1.0 cc. subcutaneous injections of toxoid at intervals of one to three months; another received two 0.5 cc. subcutaneous injections at the same intervals; a third group received 0.1 cc. twice intracutaneously, and a fourth group received 0.05 cc. twice intracutaneously. A stimulative dose was given similar to the primary dose six to twelve months after the second injection. The antitoxin content of the serums was determined.

Several of the authors' conclusions are:

1. It is safe to immunize allergic children with alum-precipitated toxoid.

2. There is no difference in antitoxin-producing ability between the allergic and the nonallergic children.

3. The intracutaneous method produces less reaction than the subcutaneous method, and the antitoxic response is sufficient to give protection.

4. A stimulating dose of toxoid given to an immunized child produces a rapid rise in the antitoxin content of the serum.

5. Active immunization has no effect on allergic manifestations.

6. Concurrent allergic therapy has no effect on the antitoxin levels produced.

HOYER, Cincinnati.

THE IN VITRO RESISTANCE OF POLIOMYELITIS VIRUS TO CHEMICAL AGENTS. EDWIN W. SCHULTZ and FRANCIS ROBINSON, J. Infect. Dis. **70**:193 (May-June) 1942.

The virus of poliomyelitis (MVA strain) exhibits a high degree of resistance in vitro to a varied array of chemical compounds and tolerates wide differences in hydrogen ion concentration. There are, however, some agents to which it is apparently highly susceptible. Their practicability has not been determined.

TOOMEY, Cleveland.

TREATMENT OF WHOOPING COUGH BRONCHOPNEUMONIA. WILLIAM P. FRANK, EDWIN F. PATTON and PAUL M. HAMILTON, J. Pediat. **20**:720 (June) 1942.

After relatively poor results were obtained with the use of hyperimmune pertussis serum and later with sulfapyridine in the treatment of bronchopneumonia associated with pertussis, Frank, Patton and Hamilton turned to the use of sulfathiazole; the mortality rate fell to 4 per cent. The blood concentration of the drug necessary for good results averaged from 2 to 3 mg. per hundred cubic centimeters of blood. The daily dose of the drug was 1 to 2 grains per pound (0.13 to 0.26 Gm. per kilogram) of body weight.

STETTENBENZ, Buffalo.

HEMOLYTIC STREPTOCOCCI RECOVERED DURING AN EPIDEMIC IN A KINDERGARTEN. PAUL L. BOISVERT and PHILIP A. BEARG, Yale J. Biol. & Med. **14**:519, 1942.

"The methods of Lancefield and of Griffith for grouping and typing hemolytic streptococci were employed in a study of a small epidemic of scarlet fever in a kindergarten.

"Thirteen of the 46 kindergarten children became ill during the epidemic. Eleven of these had scarlet fever and 2 had streptococcal cervical adenitis. Hemolytic streptococci recovered from the throats of the sick children were identified as Group A, type 3 organisms.

"Twenty-three of the 46 children had hemolytic streptococci in their throats at some time during the 4 months of study. Eighteen carried organisms of the human pathogenic group, Group A. One child had Group B organisms in his throat; another, Group C, and 3 children carried Group G strains. The streptococci from 2 of the children belonged in Group A but they were not of the epidemic type, type 3. These 7 children remained well as did their families.

"Of the 16 children who had type 3 streptococci in their throats, 13 became ill and 5 of these infected other members of their families. Three children remained well but carried the organisms to cause disease in their families. In all, 8 families (a total of 18 family members, excluding the kindergarten children) became ill with a type 3 infection.

"The families of the 23 children without hemolytic streptococci in their throats served as controls. They remained free from disease during the epidemic.

"This outbreak, though small, indicates the value of these methods in the study of epidemics."

New Haven, Conn. [AUTHORS' SUMMARY].

IMMUNIZATION AGAINST DIPHTHERIA WITH ALUM-PRECIPITATED TOXOID. F. FULTON, JOAN TAYLOR, B. MORRE, A. Q. WELLS and G. S. WILSON, Brit. M. J. **1**:315 (March 7); 349 (March 14) 1942.

Observations are carefully recorded and discussed on the reaction of a large series of subjects who had been given Schick tests before and after the administration of alum-precipitated toxoid. These results have been analyzed, and much interesting information has been obtained. Because of the great length of the article only the salient points will be brought out. They are as follows:

1. There appears to be practically no difference between the different age groups in the proportion of a weak or strong Schick reaction.

2. The percentage of positive reactions to control tests was only 1.2 per cent, and only one seventh of these occurred in children under 10 years of age. Therefore for practical purposes it seems unnecessary to make control Schick tests on children under 10 years of age.

3. It was noted that the reaction to the alum-precipitated toxoid varied somewhat with the different products used. It was also noted that 86 per cent of persons with negative Schick reactions experienced some degree of reaction while only 42 per cent of those with positive Schick reactions experienced reaction after the injection of alum-precipitated toxoid. Reactions were much more severe in older subjects than in younger.

4. The "Schick conversion rate" after two small doses of alum-precipitated toxoid, namely 0.1 and 0.3 cc., given from four to six week intervals was universally higher, ranging between 97 and 100 per cent, except for one or two batches of toxoid which when analyzed were found to have a low antigenic quality.

5. No obvious difference was discernible between the "Schick conversion rates" for boys and for girls or for children of the various age groups up to 18 years.

6. Schick tests done one year after the initial inoculation showed that the reactions were still negative in a high percentage of instances, ranging around 96 per cent. The exception to this, where the percentage fell to 74 per cent, was thought to be due to the fact that the toxoid preparation was not satisfactory.

7. Because the authors' "Schick conversion rate" in this series of tests was comparable with or better than that of other authors using larger doses of alum-precipitated toxoid, it was concluded that large doses are not necessary but that the main consideration is the choosing of a product of high antigenic potency.

BIRDSONG, University, Va.

SULPHAPYRIDINE AS AN AID TO THE POST-OPERATIVE TREATMENT OF LARYNGEAL DIPHTHERIA ASSOCIATED WITH MEASLES. D. F. JOHNSTONE, Brit. M. J. **1**:325 (March 7) 1942.

Laryngeal diphtheria associated with measles is a rather rare disease, and in the author's experience most patients with this combined condition have died of a fatal bronchopneumonia. Because of encouraging results obtained by giving sulfapyridine in the treatment of measles and its complications, this form of therapy was used for 4 children with laryngeal diphtheria associated with measles, and all of them recovered.

BIRDSONG, University, Va.

OEDEMA OF THE GLOTTIS COMPLICATING MEASLES: A REVIEW OF EIGHT PERSONAL CASES. J. H. MULVANY, Brit. M. J. **1**:638 (May 23) 1942.

In the severe and widespread outbreak of measles occurring in the winter of 1929-1930 about 950 patients with measles were seen by the author of this article and his colleagues. Of this group there were 8 with severe edema of the glottis occurring with the measles infection. Six of these patients died, and 2 recovered. One managed to survive without operation, and one survived after tracheotomy. None of the other six patients had any operations. This report was prompted by a previous report of 4 cases of measles associated with laryngeal diphtheria.

The history and course in all the cases were very similar. It is pointed out that the edema occurring in the glottis usually occurs at about the time of the appearance of the rash and is associated with a slightly croupy cough, slow dyspnea and difficulty in swallowing. When these symptoms had begun, the patients of this study rather promptly manifested extreme respiratory distress, which became severe within a relatively short time. These symptoms are to be contrasted with those of laryngeal diphtheria, in which the onset is much more insidious and the respiratory distress is gradually progressive. Difficulty in swallowing, which is one of the clearcut signs in edema of the glottis complicating measles, is not found in laryngeal diphtheria, and this symptom alone should warn the physician of the urgency of the situation. Of the 6 patients who died all died with a rapidly progressive asphyxia which occurred within a period of twenty minutes to about two hours. It is urgently advised that when edema of the glottis complicating measles is suspected the patient be under constant and competent care so that operative intervention can be started in a minimal amount of time.

In his conclusions the author states that the mortality in untreated patients is probably not far short of 100 per cent and that edema of the glottis complicating measles is not an academic curiosity but is an entity which may confront a physician during any epidemic of measles.

BIRDSONG, University, Va.

CEREBROSPINAL FLUID IN ACUTE ANTERIOR POLIOMYELITIS. ARTHUR L. EYRE-BROOK, Brit. M. J. **1**:758 (June 20) 1942.

The diagnosis of acute poliomyelitis in an infant is sometimes very difficult and not all cases of paralysis are necessarily poliomyelitis. Because of the difficulty in making a diagnosis the author calls attention to the various characteristic changes that occur in the cerebrospinal fluid. These are: "(1) an early rise in cell count, falling to normal by about the end of the second week of the illness; (2) a protein content which is almost normal until some days after the onset of paralysis, and which rises to a maximum in the third week."

Included in the article is a summary with charts of 7 cases of infantile paralysis in children admitted to the Bristol Children's Hospital. In most of these cases more than one spinal puncture was done, and it was demonstrated that the characteristic conditions were found in the spinal fluid. In the summary it is stated that the purpose of the paper is to appeal that more than one examination be made of the cerebrospinal fluid when acute anterior poliomyelitis is suspected, since by this method a more accurate diagnosis can be made.

BIRDSONG, University, Va.

APPLICATION OF HIRST'S PHENOMENON TO THE TITRATION OF VACCINIA VIRUS AND VACCINIA IMMUNE SERUM. F. P. D. Nagler, M. J. Australia **1**:281 (March 7) 1942.

Vaccinia suspensions prepared from stock virus grown on the chorioallantoic membrane of 10 day chick embryos cause specific agglutination of red cells of fowls of similar type to that observed by Hirst with influenza virus (*Science,* July 4, 1941). This agglutination is inhibited by human or animal immune serum. The method provides an accurate and convenient method of measuring the antibody response to vaccinial infection.

GONCE JR., Madison, Wis.

NOSCOMIAL TRANSMISSION OF ECZEMA VACCINATUM. ROLF BERGMAN and JONAS LINDAHL, Nord. med. (Hygiea) **11**:2714 (Sept. 20) 1941.

A 6 month old boy with eczema was admitted to a ward where 3 other infants with eczema and the healthy infants of 2 wetnurses were lying. All of the children were unvaccinated. After two days the boy's eczema showed a vaccine-like character (positive corneal reaction). His sister at home had been vaccinated three weeks previously. The boy was immediately transferred to an isolation hospital. The other 3 eczematous infants were also infected with vaccine virus, and so was also another infant with eczema from an adjacent ward. All 5 infants of the ward recovered, with remaining scars.

WALLGREN, Göteborg, Sweden.

WHOOPING COUGH WITH EXTREME GRANULOCYTOPENIA. SVEN RUDBERG, Nord. med. (Hygiea) **11**:2715 (Sept. 20) 1941.

A 9 month old girl suffering from typical pertussis (Bordet-Gengou bacilli demonstrated) fell ill with fever and displayed at the same time a granulocytotic blood picture (white cells, 8,500, with 56 per cent lymphocytes, 19.5 per cent monocytes, 3.5 per cent granulocytes and 21 per cent pathologic mononuclear cells, of which some gave positive oxidase reactions). The bone marrow gave evidence of normal erythropoiesis, while the myelopoiesis was badly

impaired, with disturbance of the maturation process. The blood picture and defects in the bone marrow slowly improved and eventually became normal. The girl recovered.

WALLGREN, Göteborg, Sweden.

DURATION OF IMMUNITY IN DIPHTHERIA VACCINATION AND SIGNIFICANCE OF REVACCINATION. KNUD BOJLÉN and INGA SCHEIBEL, Ugesk. f. læger **103**:1143 (Sept. 4) 1941.

After one injection of Ramon's anatoxin only 71 per cent of the vaccinated children acquired an antitoxin content of the blood corresponding to 1/100 unit per cubic centimeter, and this amount rapidly decreased. After a second injection four weeks later the antitoxin content was 1/100 unit per cubic centimeter in all children, and it remained at this level during one year in some but not in all. At this time a third injection was made. After these three injections the antitoxin content was satisfying in 94 per cent of the vaccinated children for at least two and one-half years. Renewed revaccinations in those who already had received the usual three injections caused a rapid increase to sufficiently high titers of the antitoxin of the blood.

WALLGREN, Göteborg, Sweden.

## Acute Infectious Diseases

A STUDY OF SEVENTY RHEUMATIC FAMILIES. A. ROSENBLUM and R. L. ROSENBLUM, Am. Heart J. **23**:71 (Jan.) 1942.

The authors found that infections of the respiratory tract preceded the onset of rheumatic fever in about half of the cases studied. In an analysis of home conditions, dampness was the only factor that could be correlated with an increased incidence of rheumatic fever. Families with rheumatic parents had a higher incidence of the disease among the siblings than those with nonrheumatic parents. Simultaneous attacks of rheumatism were frequently noted among siblings.

GIBSON, Chicago.

THE SIGNIFICANCE OF PUS CELLS IN THE SMEAR DIAGNOSIS OF GONOCOCCAL INFECTION. ARTHUR STEER, Am. J. Syph., Gonor. & Ven. Dis. **26**:191 (March) 1942.

Because the presence or absence of pus cells in diagnostic smears taken for the diagnosis of gonorrhea may be helpful to the laboratories examining such smears, it is extremely important to state the sex of the subject and the location from which the smear was taken. When the smear is taken from the urethra of men or of women, evidence of a large amount of pus is often helpful in making a diagnosis, while, at the other extreme, the finding of pus cells on a smear from the endocervix is not at all indicative of a gonorrheal infection because of the numerous other causes that may cause pus to be present at this location. Although the presence or absence of pus cells may be of some value without other evidence, their occurrence is of little significance as an indicative of gonococcic infections. In contrast, the absence of pus cells does not indicate that gonococcic infection is not present.

BIRDSONG, University, Va.

SULFADIAZINE IN EXPERIMENTAL STREPTOCOCCIC INFECTION. BARTON MCSWAIN and FRANK GLENN, Arch. Surg. **44**:231 (Feb.) 1942.

This study represents the results of the implantation of sulfadiazine into wounds of rabbits inoculated with a measured amount of culture of beta hemolytic streptococci of known virulence. Eighteen rabbits were treated by this method and 9 control rabbits were used. Though the results were not striking, the rabbits treated with sulfadiazine showed (1) a decreased mortality rate; (2) increased survival time; (3) lower percentage of positive blood cultures; (4) slightly lower percentage of positive wound cultures, and (5) apparent improvement in wound healing.

KAISER, Rochester, N. Y.

PHENOTHIAZINE IN TREATMENT OF PINWORMS. M. J. MILLER and R. D. ALLEN, Canad. M. A. J. **46**:111 (Feb.) 1942.

Phenothiazine administered in chocolate tablets was tested in 75 children infected with pinworms.

Fifty children aged 4 to 12 years were given 6 to 8 grains (0.39 to 0.52 Gm.) of phenothiazine over a period of as many days. It was effective in 64 per cent.

Twenty-three children 8 years of age were given 7 grains (0.45 Gm.) of phenothiazine over a period of three days. It was effective in all.

In 3 cases the use of phenothiazine was discontinued on the second day. These children were still infected.

Anemia was produced in about half of the children.

While phenothiazine is as effective as, or even more so than, gentian violet, the toxic effects have not been sufficiently studied to permit its recommendation for anthelmintic use in children.

MOORE, Omaha.

TETANUS TOXOID IN THE PREVENTION OF TETANUS AND SERUM REACTIONS IN ALLERGIC INDIVIDUALS. LEON UNGER, Illinois M. J. **81**:139 (Feb.) 1942.

Tetanus toxoid when given correctly prevents tetanus and the many serum reactions which have followed injections of tetanus antitoxin. Persons who are immunized by toxoid rarely need antitoxin.

The alum-precipitated toxoid is better than the plain formaldehyde-treated toxoid, because it acts more slowly and therefore stimulates antigenic response over a longer period of time. Two subcutaneous injections of alum-precipitated tetanus toxoid given at intervals of two to three months will protect almost all persons; a third dose of toxoid should be given if injury occurs after the primary injections.

Reactions from tetanus toxoid have been minor. Local reactions are rather frequent. Systemic reactions have been exceedingly rare. There is no record of any fatal reaction from tetanus toxoid. The presence of allergy is no contraindication to the use of toxoid.

When diphtheria and tetanus toxoids are given together, the resultant titers of serum antitoxin have been shown to be enhanced, the one by the other.

In the author's experience with 171 patients, local reactions occurred no more frequently in allergic than in normal persons. When a local reaction occurred, the second dose was split (0.25 cc. injected at eight weeks and another 0.25 cc. at twelve weeks). It is perfectly safe to immunize persons who have had previous injections of serum, both with and without serum sickness. Those who were allergic to horse dander can also be inoculated with tetanus toxoid with impunity.

BARBOUR, Peoria, Ill.

THE TREATMENT OF VARIOUS INFECTIONS IN CHILDREN WITH SULFATHIAZOLE. R. C. MCGAHEE, J. M. A. Georgia **31**:100 (March) 1942.

The cases of 3 children are reported, 2 with staphylococcic septicemia and 1 with influenzal meningitis, who recovered with sulfathiazole therapy. Of the 2 children with staphylococcic septicemia, 1 had an associated osteomyelitis of the right pubic bone extending into the acetabulum and involving the head of the femur. In the other the infection followed a furunculosis of the nose and face.

One and one-half to 5 grains per pound (0.09 to 0.32 Gm. per 0.5 Kg.) per day was given.

MORRISON, Savannah, Ga.

TREATMENT OF TYPE-SPECIFIC HEMOPHILUS INFLUENZAE INFECTIONS IN INFANCY AND CHILDHOOD. HATTIE E. ALEXANDER, CATHERINE ELLIS and GRACE LEIDY, J. Pediat. **20**:673 (June) 1942.

In this comprehensive paper Alexander, Ellis and Leidy present the clinical features and the treatment of three serious infections due to type B Haemophilus influenzae, namely meningitis, acute laryngotracheobronchitis and pneumonia accompanied by empyema in infants under 1 year of age.

Treatment utilizes certain biologic characteristics of Haemophilus influenzae, features strikingly similar to those of the pneumococcus. Like the latter, H. influenzae is encapsulated and gives off a specific soluble carbohydrate antigen into the body fluids. This substance can be detected by a precipitin test using diagnostic typing influenza serum against an equal quantity of body fluid containing the antigen. The amount present is indicative of the severity of the infection. Treatment is based on the administration of anticarbohydrate antibody sufficient quantitatively to neutralize the existing soluble specific carbohydrate antigen. The most efficacious antibody is in therapeutic H. influenza rabbit serum (Squibb), in which the globulin fraction has been concentrated and is expressed in milligrams of nitrogen. Treatment is successful when free antibody is present in body fluids and when added to H. influenza organisms produces capsular swelling, or the quellung reaction. This test is used in actual treatment to determine whether sufficient serum has been given.

Briefly, the therapy of influenzal meningitis is as follows:

1. Initially, a sulfonamide compound, preferably sulfanilamide (lately sulfadiazine), is given by continuous intravenous drip with 5 per cent dextrose in Ringer's solution. This speeds up

excretion of the specific soluble carbohydrate antigen and prevents further liberation of this substance by inhibiting growth of the organism.

2. Four hours later concentrated H. influenzae rabbit serum (anticarbohydrate antibody) is given intravenously. The dose is based on the spinal fluid sugar level.

3. One hour after administration of the serum the patient's serum is tested for antibody response (capsular swelling). If the response is positive, no further serum is given.

4. Administration of the sulfonamide compound is continued until recovery.

5. Further administration of serum depends on antibody response and results of examination of the spinal fluid. Alexander reports a survival of 14 of 16 patients on this regimen in 1941.

The treatment of laryngotracheobronchitis and that of pneumonia with empyema follow the same lines; serum is used if the antibody test elicits a negative response, and a sulfonamide compound is administered and other necessary measures instituted.

STETTENBENZ, Buffalo.

TREATMENT OF INFLUENZAL MENINGITIS WITH ANTI-INFLUENZAL RABBIT SERUM AND SULFAPYRIDINE. JOHN P. SCULLY and MAUD L. MENTEN, J. Pediat. **21**:198 (Aug.) 1942.

Scully and Menten report recovery in 5 of 9 cases of influenzal meningitis utilizing Squibb's anti-influenzal rabbit serum and sulfapyridine. The injection of a sodium salt of hydroxyethylapocupreine intrathecally was also used and seemed to exert some bacteriostatic effect. The importance of mastoidectomy is stressed in cases of otitic involvement.

STETTENBENZ, Buffalo.

SALMONELLA SUIPESTIFER INFECTION IN BOSTON. N. ERNEST GOULDER, MARGARET F. KINGSLAND and CHARLES A. JANEWAY, New England J. Med. **226**:127 (Jan. 22) 1942.

The organism now known as Salmonella suipestifer was isolated in 1885 by Salmon and Smith from swine with hog cholera. During World War I, suipestifer organisms were identified as the causative agents of a number of mild epidemics of acute gastroenteritis. In 1919, MacAdam reported an epidemic in Mesopotamia and Hirschfeld reported one in Serbia. Since then, many reports of similar epidemics have been published, and by 1937 Harvey had compiled a total of 1,425 cases occurring in epidemics.

The authors report 11 cases of sporadic infection due to the European type of S. suipestifer —6 patients being children, 2 newborn infants, 2 parturient women and 1 an elderly woman. In this group, 2 deaths occurred—1 in a newborn infant, who contracted both bacteremia and meningitis, and the other in the elderly woman, who acquired bacterial endocarditis.

From a survey of the literature, it is apparent that S. suipestifer infection occurs in epidemic form as acute food poisoning, with low mortality, and in endemic form at any age, with a considerable mortality, because it often complicates some debilitating disease. The endemic form is characterized by fever, frequently leukopenia, bacteremia and a tendency for the infection to localize in the bones, joints or lungs.

The bacteriologic diagnosis of this infection is discussed. A positive blood culture most frequently reveals the diagnosis, occasionally a positive stool or urine culture. S. suipestifer is identified as a paratyphoid bacillus that does not ferment arabinose.

The importance of using standard antigens and properly prepared antiserums in the identification of the various types of Salmonella is pointed out. GENGENBACK, Denver.

RESULTS OF BACTERIOPHAGE TREATMENT OF BACILLARY DYSENTERY AT ALEXANDRIA: A STATISTICAL RETROSPECT. ARTHUR COMPTON, Brit. M. J. **1**:719 (June 13) 1942.

A statistical record comparing the mortality rate of Alexandria, Cairo and the rest of Egypt including Cairo is presented. For the last twelve years bacteriophage treatment has been used in Alexandria for all patients with bacillary dysentery, but it has not been used to any extent in other parts of Egypt. It is shown that the mortality rate in Alexandria has steadily fallen from approximately 25 per cent in 1928 to 5 per cent in 1938, while that in Cairo and the rest of Egypt has varied between a maximum of 60 per cent and a minimum of 25 per cent. Since the only difference in the treatment is in the bacteriophage, it is suggested that the bacteriophage treatment is the factor which causes this difference in percentages.

A similar chart with the case mortality from typhoid is presented; the case mortality in this disease has not varied at all in the last twelve years, presumably because of no difference in treatment.

The phage treatment consists of giving an ampule of 2 cc. of bacteriophage as soon as possible and thereafter an ampule every four hours. The patient is put to bed and put on a starvation diet for twenty-four hours. Except for these differences patients in Alexandria are treated as they are in other places in Egypt. BIRDSONG, University, Va.

A CLINICAL AND THERAPEUTIC SURVEY OF AN INFLUENZA EPIDEMIC AT THE POTCHEFSTROOM MILITARY CAMP. W. EMDIN, South African M. J. **16**:101 (March 14) 1942.

Almost two and a half times as many members of the Youth Training Brigade (youths 15 to 19 years old) were affected with influenza as those of all other units combined. The epidemic occurred in two forms, mild and severe. The first phase of the severe form was often identical in symptoms and clinical signs with the benign form of the disease. The second phase of the severe form exhibited either symptoms and signs of pneumonic consolidation (patchy in distribution) or pyrexia with indefinite chest signs. The average total white cell count in each form of the disease was 12,000 per cubic millimeter, and the average percentage of polymorphonuclear leukocytes was 65 to 70. Bacteriologic findings suggested that a nonhemolytic streptococcus was probably the most important secondary invading organism. No attempt to isolate a virus was made. Sulfapyridine was used in the severe form of the disease with most satisfactory results. GONCE JR., Madison, Wis.

BRUCELLOSIS SUIS. R. G. VALLEDOR and A. HERNANDEZ, Bol. Soc. cubana de pediat. **14**:76 (Feb.) 1942.

The authors report on a case of supposed brucellosis in an 11 year old Negro child. It was the first case of that type observed in that particular Cuban province. The organism isolated bacteriologically corresponded in all its cultural characteristics to Brucella suis.

Treatment with sulfanilamide, with vaccine and finally with acriflavine hydrochloride brought about a complete cure of the condition.

The authors point out that infection with Br. suis is probably more common than is suspected and should always be considered if one is dealing with a prolonged and obscure fever. The infectious agent is easily transmitted to human beings. SCHLUTZ, Chicago.

PNEUMOCOCCIC MENINGITIS IN CHILDREN AND ITS TREATMENT. GRETA MUHL, Svenska läk.-tidning. **38**:1284 (June 6) 1941.

Muhl presents a report of 28 cases of pneumococcic meningitis in infants observed between 1926 and 1938. Most of the patients were treated with ethylhydrocupreine hydrochloride. Seven of the infants recovered. In addition 3 new cases of this condition in infants are reported in each of which recovery followed treatment with sulfapyridine.

WALLGREN, Göteborg, Sweden.

FOUR CASES OF PNEUMOCOCCIC MENINGITIS WITH RECOVERY. HENNING VOGELIUS, Ugesk. f. læger **103**:633 (May 15) 1941.

In 21 cases of pneumococcic meningitis only 4 patients survived, and these were the only ones who had been treated with large doses of sulfapyridine; 3 of these patients were in addition treated with specific serum. In two thirds of the cases the infection was of otogenic or rhinogenic origin. WALLGREN, Göteborg, Sweden.

## Chronic Infectious Diseases

A NOTE ON THE SURVIVAL OF TREPONEMA PALLIDUM IN PRESERVED CITRATED HUMAN BLOOD AND PLASMA. JOHN A. KOLMER and ANNA M. RULE, Am. J. Syph., Gonor. & Ven. Dis. **26**:156 (March) 1942.

In a single experiment a suspension of Treponema pallidum was added to citrated human blood; half of the mixture was centrifuged and the citrate-plasma-treponeme mixture and the blood-plasma-treponeme mixture were then put into a refrigerator which was kept at 4 to 6 C. The following tests were done: 1 cc. of each mixture was injected intratesticularly into rabbits at intervals of three, twenty-four, forty-eight and seventy-two hours. It was observed that the citrate-blood-treponeme mixture was infective after three, twenty-four and forty-eight hours' preservation, as evidenced by the development of a syphiloma in the testicle of the rabbit. This was not noted at the end of seventy-two hours.

The citrate-plasma-treponeme mixture was infective one and three hours after preparation as evidenced by the production of the testicular syphilomas but apparently was not infective after twenty-four, forty-eight and seventy-two hours of preservation.

It is concluded, therefore, that citrated human blood heavily infected with T. pallidum undergoes spontaneous sterilization in about seventy-two hours if it is kept at 4 to 6 C. In infected citrated plasma spontaneous sterilizations may occur in about twenty-four hours.

BIRDSONG, University, Va.

REINFECTION IN CONGENITAL SYPHILIS. SAMUEL D. ALLISON, Am. J. Syph., Gonor. & Ven. Dis. **26**:212 (March) 1942.

Two old cases of reinfection of patients who had been treated for congenital syphilis found in the files of the syphilis clinic of the Johns Hopkins Hospital are briefly reported. In addition 2 more recent cases are reported. In the discussion the author states that Halley and Wassermann "found that 97.8 per cent of all second infections in acquired syphilis occurred in individuals who were treated early in the course of their first infection. A few cases were encountered in persons whose treatment was postponed until the early latent stage," and in no instance did reinfection occur in these patients. "It appears that second infections in congenital syphilis may occur not only in patients whose treatment was not begun until comparatively late but also in those inadequately treated in early infancy who later experienced clinical recurrences which necessitated resumption of treatment. These observations suggest the possible difference between the immunity developed by congenitally syphilitic individuals and that which develops in those who do not become infected until after birth."

Three of the 4 patients here reported on were girls within or close to the child-bearing period, at which time they contracted syphilis a second time. Had their reinfection not been detected and treated and had any of them become pregnant shortly thereafter, they would in all probability have given birth to syphilitic children who would have been erroneously considered as good examples of third generation syphilis. Because of the rather universal treatment of congenital syphilis in the recent past, it is to be expected that the incidence of superinfection will increase, as large numbers of children so treated are now approaching the child-bearing stage.

BIRDSONG, University, Va.

OSSEOUS SYPHILIS: CONFUSION IN DIAGNOSIS; A REPORT OF TWO CASES OF OSTEOMYELITIS OF LATE SYPHILIS. LEE J. ALEXANDER and ARTHUR G. SCHOCH, Am. J. Syph., Gonor. & Ven. Dis. **26**:397 (July) 1942.

Two cases are reported in which osseous syphilis was confused with other diseases. In the first case a Negro aged 46 entered the hospital with the complaint of headache of about a year's duration associated with stoppage in the nose. A diagnosis of sinusitis was made. Roentgen examination of the sinuses and skull revealed pronounced destruction of the skull. The serologic reaction of the blood at this time was positive for syphilis but by a clerical error was reported as negative on this patient's chart, which confused the picture. The patient later came back to the hospital. Because of the unusual roentgen findings, examination for Paget's disease and for carcinoma was done. At this time another serologic test was taken, which gave a strongly positive reaction. The results of all tests for Paget's disease and for carcinoma were negative. The patient made a dramatic recovery after antisyphilitic treatment was begun.

The second instance reported was that of a 19 year old Negro girl who complained of feeling tired and lazy and rather suddenly experiencing a severe pain in her neck. Roentgen examination revealed evidence of a destructive lesion of the cervical vertebrae and also of several of her ribs. The serologic reactions of her blood were positive. The condition was diagnosed as tuberculosis, and the patient was treated along these lines. She did not improve and continued to have fever, and because she did not improve with the measures employed it was decided to give a therapeutic test for syphilis. This patient's temperature immediately dropped to normal after a couple of treatments, and her pain disappeared even before there were any roentgen changes in her bones. With adequate antisyphilitic treatment her recovery was complete.

In the conclusion the authors state that when unusual clinical findings which are difficult to explain arise and syphilis is suspected it is wise to check negative serologic reports and when osseous lesions present a picture that makes the diagnosis a problem one is justified, especially with a positive serologic reaction, in making therapeutic trial of antisyphilitic drugs.

BIRDSONG, University, Va.

IMMUNITY IN TUBERCULOSIS. F. M. POTTENGER, Am. Rev. Tuberc. **45**:651 (July) 1942.

Attempts that have been made to separate the various phases of immunity have tended to obscure rather than clarify the real nature of the process and have led to confusion rather than to understanding. Some speak of allergy in contradistinction to immunity. This is illogical. It should be considered as being only one phase of tissue sensitization, while immunity includes all specific protective phenomena. Immunity in its widest sense may be defined as an altered reaction of the host necessary to protect it from bacteria. The preponderantly exudative process usually termed allergic may be considered as an early, less complete phase of the total immunity response to an infection. Immunity is always a healing and protective phenomenon but one which, to be efficient, must be built up *pari passu* with the infection against which it protects.

Man's normal physiologic response is sufficient to protect him against mild infections; a heightening of these same physiologic forces occurs as immunity. When bacilli enter the tissues, they attempt to destroy the local cells and to multiply. The cells of the body react to destroy the bacilli. The bacillary fractions set free as a result of this struggle sensitize the tissues to further contact with bacillary products. If the infection is not too great, these sensitized tissues develop enhanced destructive powers against invading bacteria; in the case of tuberculosis a more rapid and extensive tubercle formation results.

Immunity in tuberculosis starts to develop as soon as the infecting bacilli begin to multiply and the bacillary products in minute quantities begin to make their escape into the tissues. In primary infections this protection seems to be inadequate and the bacilli rapidly find their way into the regional lymph nodes and often beyond. As the body cells become sensitized to the bacillary protein (tuberculin), reaction occurs, at first as an inflammatory exudative reaction with an outpouring of wandering cells and accompanied by proliferation. As immunity increases, the cells become less sensitive and the inflammatory reaction less prominent, while the bacilli are inhibited in their multiplication and growth and proliferation becomes more evident.

The allergic, inflammatory exudative reaction is particularly characteristic in adolescence and young adult life. Here the chief factor in defense is the seizing and anchoring of bacilli by inflammation and tubercle formation. In persons with chronic ulcerative tuberculosis, on the other hand, the body tissues must be able to combat frequent episodes of reinfection and prevent the multiplication and spread of vast numbers of bacilli if the organism is to survive. In such cases the host may be assumed to have acquired a relative desensitization together with a marked ability to inhibit growth and multiplication and an increased power to destroy bacilli, as a result of many reinoculations of small numbers of organisms.

Immunity is essential to healing but cannot produce this result unless proper compensation can take place between the pulmonary tissues and the thoracic cage. This may be brought about naturally by (1) emphysema, (2) shifting of the mediastinum, (3) elevation of the diaphragm and (4) contraction of the bony cage and operatively by (1) pneumothorax, (2) operations on the phrenic nerve, (3) severing of adhesions and (4) thoracoplasty, but immunity must heal the lesion.

Since bacillary proteins (tuberculin) are factors in creating immunity and stimulating healing, there is a logical basis for the therapeutic use of these substances obtained from dead bacilli. Properly used, tuberculin will produce many of the effects which are necessary to healing, though it is not a perfect immunizing agent. EUGENE SMITH, Ogden, Utah.

DIFFERENTIAL DIAGNOSIS IN PULMONARY TUBERCULOSIS. JAMES M. WARING, Am. Rev. Tuberc. **45**:666 (June) 1942.

This paper should be read by all physicians in general practice as well as by those in all the specialties. Waring reviews the old established and the more recent diagnostic procedures related to the diagnosis of this protean disease. "The time to suspect tuberculosis is when the patient first puts his foot in the door of the doctor's office." From this point he proceeds to discuss the symptomatology of the disease, stressing the point that the appearance of good health, a feeling of well-being and even the absence of physical signs do not exclude pulmonary tuberculosis. In differential diagnosis special mention is made of coccidioidomycosis of the lung, which, while very rare outside of California and Arizona, bears such a remarkable resemblance to tuberculosis, pathologically, clinically and roentgenologically, that it is important to be familiar with its manifestations.

In obscure cases, a differential diagnosis of pulmonary disease may not be possible without repeated examinations of the chest, roentgen examinations and bronchoscopic examinations, and also culture, gastric lavage and guinea pig inoculation.

EUGENE SMITH, Ogden, Utah.

TUBERCULOSIS OF THE MEXICAN PLATYFISH (PLATYPOECILUS MACULATUS). JAMES A. BAKER and WILLIAM A. HAGAN, J. Infect. Dis. **70**:248 (May-June) 1942.

A naturally occurring outbreak of tuberculosis in the small tropical platyfish, Platypoecilus maculatus, was studied. The causative organism was isolated and studied, and the authors describe it as fully as an organism of the acid-fast group can be described. The biologic characteristics and pathogenic properties indicate that this organism is a pure parasite and pathogen.

A second acid-fast organism was repeatedly isolated from the water of the aquariums in which diseased and normal platyfish were kept. Attempts to infect platyfish and goldfish with this organism failed when large amounts of culture were fed; however, when parenterally inoculated, the culture killed the goldfish. Since several acid-fast organisms which are regarded as purely saprophytic did the same thing, it is concluded that inoculation into the tissues is not a good way to determine pathogenicity of such cultures.

TOOMEY, Cleveland.

A TUBERCULOSIS SURVEY OF HOUSEHOLD GROUPS. CHESTER A. STEWART, J. Pediat. **20**:711 (June) 1942.

In an extensive search for tuberculous infection in his private practice, Stewart has surveyed by means of roentgen examinations and Mantoux tests 4,251 patients (2,161 children and 2,090 adults). The initial study revealed positive reactions to tuberculin in 3.2 per cent of the children and 55 per cent in adults. Further examination uncovered 2 persons with active pulmonary tuberculosis and 32 additional persons with tuberculosis to whom members of their families had been exposed.

Follow-up study of 654 children who had reacted negatively to the Mantoux test three years previously showed that 2.6 per cent had since acquired positive reactions. Through further investigation of these 17 children, 6 persons with active pulmonary tuberculosis were discovered.

This study demonstrates the importance of conducting a tuberculosis survey in a physician's private practice.

STETTENBENZ, Buffalo.

LEPRA NERVOSA IN A GIRL FIVE YEARS OLD WITH NEURITIS OF THE CUBITAL NERVE. T. VALLADOR, L. EXPOSITO, J. PEDRERA and R. FUSTE, Bol. Soc. cubana de pediat. **14**:372 (July) 1942.

This child showed contracture of the fourth and fifth fingers of the left hand with atrophy of the hypothenar group of muscles and the muscles innervated by the cubital nerve in the same arm.

The family history was noncontributory. The present illness began six or seven months before the child's admission to the hospital with a deep ulceration localized in the hypothenar eminence of the left hand. It was not painful and was unaccompanied by fever. Several months later the ulcer healed, but a scar was left which is noticeable at present.

An area of anesthesia was found in the region of the scar. The cubital nerve along the arm was increased in size. In the skin of the posterior region of the left arm anesthetic macules were found. Pathologic examination showed them to be lesions of a form of leprosy.

SCHLUTZ, Chicago.

PULMONARY TUBERCULOSIS IN CHILDREN. SIGRID HOLM and V. POULSEN, Nord. med. (Hospitalstid.) **10**:1462 (May 10) 1941.

Holm and Poulsen report on 669 children suffering from pulmonary tuberculosis. Six hundred and thirty-two of them reacted to a percutaneous tuberculin test, the remaining 37 needed intracutaneous tests with 0.1 to 1 mg. of tuberculin in order to react positively. Six hundred and fifty children were examined for tubercle bacilli in the stomach, and in 520 of them bacilli were demonstrated, more often in infants and younger children than in older ones. Of 88 tuberculous infants, 38 died, 2 were still sick, 44 were healthy and 4 could not be found. For 113 children 1 to 2 years of age, the corresponding figures are 23, 9, 74 and 7. Of 321 children 2 to 7 years of age, 20 died, 17 were still sick, 267 were healthy and 17 could not be found. Of 147 older children, of 7 to 14 years of age, 5 died, 20 were still sick, 112 were healthy and the condition of 10 was unknown.

WALLGREN, Göteborg, Sweden.

CALMETTE VACCINATION OF SCHOOL CHILDREN. ERIK TÖRNELL, Nord. med. (Hygiea) **11**: 2768 (Sept. 27) 1941.

In order to increase the specific resistance to tuberculosis in children who at puberty are finishing their school training, Törnell has vaccinated them with BCG. In all, 914 children have been examined, of whom 636 reacted negatively to tuberculin, and of them 557 (86 per cent) consented to BCG vaccination by intracutaneous injection. The author advances the opinion that primary and early postprimary tuberculosis after virulent infection are prevented by this procedure.

WALLGREN, Göteborg, Sweden.

METHOD AND PRELIMINARY RESULTS OF BCG VACCINATION. EYOLF DAHL, G. HERTSBERG and ERLINE REFSUM, Nord. med. (Norsk mag. f. lægevidensk.) **10**:1381 (May 3) 1941.

The authors report on the vaccination with BCG of 2,354 persons of Oslo, Norway, during the years 1928 to 1940. Only persons whose reaction to tuberculin was negative were vaccinated, and the vaccine was applied intracutaneously. Vaccines prepared at different times were of different effectivity. With a good vaccine 98 per cent of the persons vaccinated became sensitive to tuberculin two months after vaccination. The vaccine allergy may disappear after six months, but 96.8 per cent of the subjects were still allergic after four years. The frequency of tuberculosis in vaccinated subjects amounted on the average to only one half of the frequency in nonvaccinated persons of corresponding age groups in Oslo. A closer scrutiny of the cases of tuberculosis showed that only in 9 could the illness be regarded as due to an unsuccessful BCG vaccination. The authors emphasize the necessity of testing the vaccinated persons with tuberculin and examining them roentgenologically at regular intervals.

WALLGREN, Göteborg, Sweden.

## Diseases of Blood, Heart and Blood Vessels and Spleen

COARCTATION OF THE AORTA IN CHILDREN. S. P. SCHWARTZ and D. GREENE, Am. Heart J. **23**:99 (Jan.) 1942.

Two children were studied who exhibited signs of coarctation of the aorta involving the origin of the left subclavian artery. Blood pressure was not obtainable in the left arm or in the legs. Both children showed asymmetric development of the upper half of the body. The right half of the chest was larger than the left, and the right arm was longer and larger than the left arm.

GIBSON, Chicago.

RELATIONSHIP BETWEEN THE SPLEEN AND THE MORPHOLOGIC PICTURE OF BLOOD REGENERATION. W. O. CRUZ and F. S. ROBSCHEIT-ROBBINS, Am. J. M. Sc. **203**:28 (Jan.) 1942.

Detailed morphologic examinations of the blood cells of 3 splenectomized and 19 nonsplenectomized dogs were made.

In the splenectomized dogs made anemic by bleeding or by injections of acetylphenylhydrazine, the number of normoblasts in the peripheral blood during the first phase of the regenerative period is four times greater than in the nonsplenectomized animals. In some instances the number may be even higher, attaining 70,000 normoblasts per cubic millimeter. Furthermore, the number of primitive erythroblasts (basophilic erythroblasts of Ferrata) is much higher in the splenectomized animals, in some cases reaching about 40 per cent of the nucleated red cells in the circulation.

These findings suggest a relation between a function of the spleen and the maturation of the erythroblasts in the bone marrow or a factor acting on the launching mechanism of new red cells into the general circulation.

HENSKE, Omaha.

THE HEART IN SICKLE CELL ANEMIA. HARRY F. KLINEFELTER, Am. J. M. Sc. **203**:34 (Jan.) 1942.

No cause other than the profound anemia is found to explain the cardiac changes in patients with sickle cell anemia. The mechanism by which any anemia produces changes in the heart is not entirely understood, but it seems probable that the hypertrophy and dilatation are compensatory for the prolonged anoxemia.

The cardiac changes of patients with sickle cell anemia are more pronounced than the changes found in other anemias. This is because sickle cell anemia is somewhat unique in the long duration of such a severe degree of anemia.

The prolonged auriculoventricular conduction time is probably due to increased vagal tone, which also appears to be secondary to, or compensatory for, the prolonged anoxemia.

HENSKE, Omaha.

THE CLINICAL VALUE OF STERNAL BONE MARROW PUNCTURE. THEODORE H. MENDELL, DAVID R. MERANZE and THEODORE MERANZE, Ann. Int. Med. **16**:1180 (June) 1942.

The aspiration of bone marrow is a safe and adequate method for the study of many diseases. It is essential in the diagnosis and prognosis of diseases of the blood-forming organs and should be a part of the study of any hematologic problem. The authors' experience is based on 300 sternal punctures.

READING, Galveston, Texas.

A PATHOLOGICAL STUDY OF THE SIGNIFICANCE OF THE SYSTOLIC MURMUR. M. S. REDNICK, Ann. Int. Med. **16**:1197 (June) 1942.

In an effort to determine the significance of the systolic murmur, the author examined 1,148 necropsy reports, of all deaths with necropsy occurring in a hospital over a period of five years. In this group 23.1 per cent were reported to have had one or more systolic murmurs, prior to death. In only 3.7 per cent of these could no clinical or pathologic explanation for the murmurs be found.

READING, Galveston, Texas.

CONGENITAL HEART DISEASE MANIFESTING ARRHYTHMIA IN UTERO. NATHAN M. FENICHEL, New York State J. Med. **42**:151 (Jan. 15) 1942.

One month before delivery a routine examination disclosed an unusual irregularity of the fetal heart sounds. The arrhythmia persisted until the birth of the child. An electrocardiogram taken on the fifth day of life was interpreted as demonstrating blocked auricular extrasystoles. The arrhythmia continued with alternating periods of regular heart beats for a year. At the end of two months a harsh, grating systolic murmur accompanied by a thrill appeared over the pulmonic area and over the sternum in the third interspace. Later a precordial bulge was apparent, and a definite diagnosis of congenital heart disease was made.

AIKMAN, Rochester, N. Y.

PERNICIOUS ANEMIA. J. PINHOLT ANDREASEN, Ugesk f. læger **103**:783 (June 12) 1941.

Andreasen reports a case of hyperchromic, megaloblastic pernicious anemia with leukopenia and lymphocytosis and achylia in a 14 year old girl. The blood picture and the sternal marrow were typical. There was an immediate reaction to treatment with liver, and the sternal punctures made several times during the course of treatment showed the changes characteristic of pernicious anemia during treatment with liver.

WALLGREN, Göteborg, Sweden.

## Diseases of Nose, Throat and Ear

MANAGEMENT OF CAVERNOUS SINUS THROMBOPHLEBITIS. GORDON D. HOOPLE and IRL H. BLAISDELL, Ann. Otol., Rhin. & Laryng. **50**:503 (June) 1941.

The discussion is confined to the anterior fulminating type of thrombophlebitis of the cavernous sinus. It is usually due to infection of the sinus through the ophthalmic veins, occasionally by way of the pterygoid plexus. The acute fulminating type differs considerably from the "chronic compensatory" type, which results from retrograde thrombosis from vessels communicating with the cavernous sinus. In many cases of the latter type there is only thrombosis, and not thrombophlebitis, and the clot may be sterile. Most cases of the fulminating type follow lesions about the upper lip and nose, and the organism is usually the staphylococcus. The criteria for diagnosis are: (1) a known site of infection; (2) evidence of infection of the blood stream; (3) early signs of venous obstruction in the retina, conjunctiva or eyelid; (4) paresis of the third, fourth and sixth nerves, resulting from inflammatory edema; (5) formation of an abscess in the neighboring soft tissues, and (6) evidence of meningeal irritation.

The authors report 2 cases, in 1 of which the patient recovered. They plan the future management of patients accordingly: (1) Start treatment early; (2) discard ligation; (3) seek ophthalmologic consultation early and often, even as often as every three hours; (4) determine the causative organism and give the appropriate drug of the sulfanilamide group (if the causative organism cannot be determined, use sulfathiazole [2-(paraaminobenzenesulfonamido)-thiazole], as most infections are staphylococcic); (5) with the drug give heparin, in large doses at first to reduce the coagulation time as soon as possible and then in regulated doses to maintain a two hour level for the coagulation time; (6) watch the blood, urine and spinal fluid for evidence of infection and treat accordingly, and (7) operate if the patient's progress is unsatisfactory and surgical treatment is indicated.

M. V. MILLER, Philadelphia [ARCH. OTOLARYNG.].

ORBITAL CELLULITIS. L. J. WALLNER, Ann. Otol., Rhin. & Laryng. **51**:127 (March) 1942.

Orbital cellulitis is an acute inflammation of the soft tissues of the orbit, characterized by redness and edema of the lids, proptosis, limitation of the globe, chemosis of the conjunctiva and a septic temperature. The veins are important. The superior and inferior ophthalmic veins drain the area supplied by the ophthalmic artery; the superior ophthalmic vein receives the anterior and posterior ethmoid veins and has a large communication with the facial system through the angular vein; the inferior ophthalmic vein communicates with the pterygoid plexus; the two ophthalmic veins do not follow the artery out of the orbit but leave through the superior orbital fissure to enter the anterior wall of the cavernous sinus. There are no lymphatics or nodes in the orbit. In 70 to 80 per cent of cases the condition follows acute sinusitis—frontal sinusitis in adults and ethmoiditis in children. It is much more frequent in infants and young children than in adults. Trauma from penetrating wounds or foreign bodies in the orbit may be the cause. The pathologic change may be a true cellulitis of the soft tissues, progressing to abscess, a subperiosteal abscess between the periosteum and the bone of the orbit or thrombophlebitis of the orbital veins. The author believes that venous stasis is important in producing the clinical picture and cites experiments to indicate this. Differentiation of orbital cellulitis and thrombosis of the cavernous sinus may be difficult or impossible. Conservative treatment will be sufficient in many cases of cellulitis.

M. V. MILLER, Philadelphia. [ARCH. OTOLARYNG.]

ANALYSIS OF SEVEN HUNDRED AND SIXTY-FIVE TONSILLECTOMIES BY THE SLUDER TECHNIC. RENE ESPINOSA, Rev. otorrinolaring. **1**:87 (March) 1942.

Espinosa advocates the Sluder procedure in all cases of tonsillectomy. In this series of 765 cases there were only 3 instances of hemorrhage. He states that there is often some residual tonsillar tissue, usually at the inferior pole, that must be removed by a snare wire.

PERSKY, Philadelphia. [ARCH. OTOLARYNG.]

TREATMENT OF SUPPURATIVE OTITIS OF MORE THAN THIRTY DAYS' DURATION. ERIK L. HOFFMANN, Nord. med. (Hospitalstid.) **10**:1599 (May 24) 1941.

Of the 128 patients, 6 were adults, and in these bad results were shown. All the remaining 122 patients were children, of whom 13 had to be operated on at a later date. Of the latter, 2 died and 11 were discharged with satisfactory results. One hundred and nine patients had received only conservative treatment and were discharged after more than thirty days (average seventy days); 90 per cent of them had satisfactory results; 7 per cent had less satisfactory results, and 3 per cent died. The advantage of the conservative method of treatment is the infrequent need for operation (11.7 per cent of cases); the disadvantage is the long and expensive stay at the hospital.

WALLGREN, Göteborg, Sweden.

TONSILLITIS AND STOMATITIS CAUSED BY VACCINE VIRUS. GUNNAR ÖRN, Nord. med. (Hygiea) **11**:2714 (Sept. 20) 1941.

An unvaccinated girl of 4 years whose brother had been vaccinated with smallpox vaccine nineteen days previously fell ill with high fever and rapidly increasing pustules on the tongue and on the tonsils. The corneal reaction was positive. The child recovered.

WALLGREN, Göteborg, Sweden.

## Diseases of Lungs, Pleura and Mediastinum

BRONCHOPNEUMONIA OF UNKNOWN ETIOLOGY IN A GIRLS' SCHOOL. WORTH B. DANIELS, Am. J. M. Sc. **203**:263 (Feb.) 1942.

The author reports on an outbreak of bronchopneumonia in a girls' school that occurred between Nov. 25, 1940, and Jan. 7, 1941, and affected 13 of 90 students, an incidence of 14.4 per cent. Previously reported outbreaks have been largely among males. The illness began, as a rule, abruptly with headache, mild sore throat and fever. This was shortly followed by a characteristic dry, harsh, nonproductive cough. Abnormal pulmonary conditions were absent early and even later in the disease were less than would be expected in patients having definite infiltration of the lung. Reduced or normal leukocyte counts were present, and the sputum contained a mixed flora. Roentgenograms of the chest usually showed an infiltration of moderate density beginning at the hilus and later extending in a fanlike fashion toward the

periphery of the lung. This was usually limited to one lobe. The disease was mild and was rarely accompanied by toxemia. Fever persisted for from one to fourteen days. There were no deaths. No virus or rickettsia was obtained by animal inoculations.

HENSKE, Omaha.

PREOPERATIVE AND POSTOPERATIVE INFECTIONS OF THE RESPIRATORY TRACT IN RELATION TO INHALATION AND SPINAL ANESTHESIA. JOHN LYFORD III, Arch. Surg. **44**:41 (Jan.) 1942.

The report deals with the relative incidence of acute infections of the respiratory tract after ether, cyclopropane and spinal anesthesia in patients who had been shown to have the kind of low grade, chronic preoperative infections of the respiratory tract not generally considered by surgeons to be contraindications to even elective operations. From a study of 120 cases in which abdominal operations were performed it was noted that acute postoperative infections of the respiratory tract occurred two and one-half times as frequently after spinal as after inhalation anesthesia. For all types of anesthesia postoperative infections of the respiratory tract developed more frequently in the patients who had low grade preoperative infections of the respiratory tract than in a similar series in whom such preoperative infections did not exist.

KAISER, Rochester, N. Y.

THE TREATMENT OF PNEUMONIA IN INFANTS AND CHILDREN. C. M. BURPEE and W. A. WILKES, J. M. A. Georgia **31**:90 (March) 1942.

This is a résumé of the treatment of pneumonia in infants and children. The authors recommend the following procedures:

1. Early use of a sulfonamide compound, the preparation being determined by the type of organism present.

2. Use of a specific serum in conjunction with the sulfonamide compound for critically ill patients or those who fail to respond to chemotherapy.

3. Transfusions in cases of streptococcic and staphylococcic pneumonia.

4. Administration of oxygen and initiation of symptomatic measures to increase the comfort of the patient and afford much needed rest.

MORRISON, Savannah, Ga.

LUNG CYSTS. W. W. ANDERSON, South. M. J. **35**:216 (Feb.) 1942.

A large number of pulmonary cysts occur during the first year of life. The author believes that their origin is commonly embryologic.

If the pressure within the cysts is great enough and the walls are correspondingly weak enough, the cyst ruptures. It may rupture into the pleural cavity and cause an empyema. The cyst may rupture into a bronchus and become partially or completely collapsed.

The diagnosis of air-expansive pulmonary cysts is usually made by roentgen examination. Air cysts in the lung may occur late in pneumonia. All children with pneumonia should have a roentgen examination before they are dismissed from the hospital.

The most successful treatment is operative and consists in partial lobectomy.

SCHLUTZ, Chicago.

A CASE OF OBSTRUCTIVE LOBAR PULMONARY EMPHYSEMA IN AN INFANT. AXEL BIERING, Nord. med. (Hospitalstid.) **10**:1375 (May 3) 1941.

The clinical features of the case were cyanosis and expiratory dyspnea, which were intensively aggravated when the child cried, and displacement of the heart to the left. There was a permanent positive pressure in the emphysematous lobe. The cause of the obstruction was obscure but was thought to be a bronchial malformation.

WALLGREN, Göteborg, Sweden.

STUDIES ON PNEUMOCOCCUS ANTIBODY PRODUCTION IN CHILDREN. J. CLAUSEN and EMILIE FABER, Nord. med. (Hospitalstid.) **10**:1757 (June 7) 1941.

The authors investigated the production of antibodies in 43 children suffering from lobar pneumonia and bronchopneumonia. Actively produced antibodies were found in 55 per cent. The younger the patient, the less is the ability to produce antibodies. From birth to 2 years of age only 1 of 11 children examined produced antibodies; at 2 to 3 years of age 4 of 6 and at 3 to 11 years of age 15 of 19 persons examined showed production of antibodies. Antibodies were produced in 11 of 16 patients with lobar pneumonia and in 4 of 13 patients with bronchopneumonia. On an average the antibodies appeared on the seventh or eighth day in

children treated with a sulfonamide compound as well as in untreated children. In some cases antibodies were demonstrated up to eighty-five days after illness; in other cases, only for a few days.

WALLGREN, Göteborg, Sweden.

## Diseases of the Gastrointestinal Tract, Liver and Peritoneum

FAMILIAL OCCURRRENCE OF CHRONIC ULCERATIVE COLITIS (THROMBO-ULCERATIVE COLITIS): REPORT OF CASES. RAYMOND J. JACKMAN and J. ARNOLD BARGEN, Am. J. Digest. Dis. **9**:147 (May) 1942.

Eight families are reported in which there were 2 or more members with thromboulcerative colitis. In seven families, there were 2 members affected. In one family there were 3 members affected, who were examined at the Mayo Clinic. This family history revealed 2 other members who had symptoms of chronic ulcerative colitis, 1 of whom died of the disease.

The authors do not think chronic ulcerative colitis is familial or infectious, since the incidence of the disease occurring in more than 1 member of a family in a group of 900 cases is only 1.8 per cent.

MORRISON, Savannah, Ga.

THE EFFECT OF MAGNESIUM SULPHATE UPON THE SPHINCTER ODDI OF MAN. GEORGE S. BERGH and JOHN A. LAYNE, Am. J. Digest. Dis. **9**:162 (May) 1942.

This experimental study was carried out on patients who previously had undergone cholecystectomy, choledochotomy and intubation of the common bile duct. As a preliminary procedure, the anatomic status of the bile ducts was demonstrated to be normal by cholangiographic examination. Several weeks or months intervened between operation and the time of the experiments. Studies were carried out after the patients had fasted for eight hours.

Details of the experiment are described. The conclusions were: 1. Magnesium sulfate may produce any one of four types of effects on the sphincter of the common bile duct. It may produce relaxation, with or without an initial contraction, followed by a return to the original tonus level, or there may be no effect. 2. The drug is equally effective in producing relaxation of the sphincter of Oddi whether administered orally or intraduodenally. Relaxation, however, is not constant and is considerably less than relaxation following the administration of amyl nitrate or the ingestion of a fatty meal. The average maximum decrease in sphincter resistance in those subjects showing relaxation following the administration of magnesium sulfate is slightly less than 4 cm. of saline solution pressure.

MORRISON, Savannah, Ga.

COMPLETE CICATRICAL STENOSIS OF THE ESOPHAGUS: PERMEATION MADE POSSIBLE BY EXTERNAL OPERATION IN CERTAIN CASES. FLETCHER D. WOODWARD, Ann. Otol., Rhin. & Laryng. **51**:94 (March) 1942.

Woodward points out that of the cases of accidental injury, those in which the lesion was due to ingestion of a caustic through error, the stricture was in the upper third of the esophagus in 53 per cent, in the middle third in 24 per cent and in the lower third in 24 per cent. Of the cases in which the lesion developed after attempted suicide, the stenosis occurred in the middle third in 71 per cent and in the upper and lower thirds in 11 per cent each. This, he says, is due to the deliberate swallowing of a larger amount of the caustic. The upper third is most accessible to the external surgical approach, but any part may be approached by modern surgical means.

The emergency treatment of the acute condition is prompt lavage of the face, mouth, esophagus and stomach with the proper neutralizing solution. A rubber feeding tube is inserted through the nose to the stomach and left in place for from ten to fourteen days. Roentgenographic or esophagoscopic studies are not made for three weeks. If there is beginning stricture, careful and gentle bougienage is carried out through the esophagoscope.

Most patients appear from two to four months after ingestion of the caustic, when they begin to have difficulty in swallowing, with pain and loss of weight. They are studied after ingestion of an opaque meal and then with an esophagoscope. If the stricture is not too severe, dilatation is carried out with the Jackson flexible bougies under visualization. If the stricture is pronounced, the patient is referred to the surgeon for two stage gastrostomy, as the sooner the patient can receive proper food and liquids the earlier the more active treatment can be started. Gastrostomy should be done with due consideration to its type

and location for future retrograde bougienage. When the gastrostomy wound is healed, an attempt is made to have the patient swallow a string. The free end is then fished out of the stomach and tied to the upper end on the outside. When the string is in place, future treatment is carried out by the retrograde method of Tucker. Usually treatment is necessary for about two years before the gastric opening may be closed, and even after that dilation may be necessary.

If these measures fail, the patient must keep the gastrostoma for life, run the risk of perforation by bougienage or undergo surgical treatment. The external route for the operation is advocated, and cases are reported, with the method used in each.

M. V. MILLER, Philadelphia. [ARCH. OTOLARYNG.]

DIFFICULTIES IN THE ENZYMIC TITRATION OF DUODENAL CONTENTS. WILBURT C. DAVISON, Bull. Johns Hopkins Hosp. **70**:504 (June) 1942.

The author discusses twelve factors which are often responsible for the inaccuracies in the reports of the enzymic activity of duodenal contents.

LYTTLE, New York.

STUDIES ON THE ANTIBACTERIAL FACTORS OF HUMAN SALIVA. MARY VAN KESTEREN, BASIL G. BIBBY and GEORGE PACKER BERRY, J. Bact. **43**:573 (May) 1942.

The authors found in previous studies that the growth of 110 of 169 strains of bacteria tested was prevented by human saliva. This effect was more pronounced against some types of organisms than against others, those most affected being bacilli and cocci isolated from air and water; those least affected were organisms isolated from the mouth. Differences occurred in the inhibitory potency of saliva from subject to subject and from time to time. In the present study an attempt was made to establish more definitely whether there is more than one antibacterial agent in saliva and to investigate the properties of such agents. Repeated experiments gave results which appeared to show that bacteria tested fall into two groups in respect to the action of saliva. In the first group are Micrococcus lysodeikticus, Sarcina lutea and Bacillus megatherium; in the second group, Staphylococcus aureus, Streptococcus haemolyticus, Lactobacillus acidophilus, Bacillus subtilis and the enteric organisms. This has suggested the existence of two antibacterial agents in saliva.

STOESSER, Minneapolis.

CHEMOTHERAPY IN THE TREATMENT OF INFECTIOUS DIARRHEA IN CHILDREN. M. HINES ROBERTS, J. M. A. Georgia **31**:94 (March) 1942.

This is a report from the Henrietta Egleston Home for Children in Atlanta, Ga., where 24 patients with infectious diarrhea were treated with sulfathiazole during 1940. The results are compared with those obtained on patients with infectious diarrhea treated during an eleven year period preceding 1940, and they compare favorably.

Adequate dosage is thought to be between 1 and 1½ grains per pound (0.65 to 0.13 Gm. per 0.5 Kg.) of body weight per day. The author feels that many of the so-called non-infectious diarrheas are really infectious in origin.

MORRISON, Savannah, Ga.

THE CHEMOTHERAPY OF INFANTILE DIARRHEA: A COMPARISON OF SULFATHIAZOLE AND SULFAGUANIDINE. RICHARD B. TUDOR, J. Pediat. **20**:707 (June) 1942.

Tudor treated 31 patients with diarrhea who were suffering from bacillary dysentery and parenteral diarrhea with sulfathiazole and sulfaguanidine. Of the group 16 received sulfathiazole and 15 received sulfaguanidine. Results, which were excellent, indicated that the two drugs are equally effective.

STETTENBENZ, Buffalo.

GASTROINTESTINAL ALLERGY AND THE CELIAC SYNDROME. RALPH H. KUNSTADTER, J. Pediat. **21**:193 (Aug.) 1942.

Kunstadter describes 3 children who presented the typical picture of celiac disease but who failed to respond to the usual diet for that disease. Study revealed multiple food allergies in all 3 infants. Removal of cow's milk from the diet resulted in cessation of the diarrhea and progressive gain in weight. The author believes that gastrointestinal allergy should be considered in any case of chronic diarrhea, particularly if idiosyncrasy to certain food is present, and especially in children with a positive allergic family history.

STETTENBENZ, Buffalo.

CHOLECYSTITIS AND CHOLELITHIASIS IN A CHILD. C. BISSON, Union méd. du Canada **71**:140 (Jan.) 1942.

A girl of 9 years began to have abdominal pain with vomiting. After three or four hours the symptoms lessened, and after a week they had practically ceased. Then suddenly the patient began to vomit once more and complained of pain which was not localized. Although she was moderately constipated, the stools were not clay-colored. Her temperature was 101.3 F.; her white blood cells were increased to 24,400, of which 90 per cent were polymorphonuclear leukocytes. Her abdomen was acutely tender over the right upper quadrant. The preoperative diagnosis was appendical abscess beneath the liver. The operation revealed a scarred appendix and an acute cholecystitis with numerous small stones. Convalescence was uneventful.

DAFFINEE, Boston.

APPENDICITIS IN EARLY INFANCY. R. C. AGUIRRE, S. D. ALZAGA and D. A. GIRALDES, Arch. argent. de pediat. **13**:3 (July) 1942.

The author believes that in acute appendicitis in early infancy the child should be operated on the moment the condition is diagnosed, regardless of the actual state of the infant. It is dangerous and often fatal to defer the operation too long.

SCHLUTZ, Chicago.

ACUTE APPENDICITIS IN SMALL CHILDREN. EINAR EDBERG, Svenska läk-tidning. **38**:1118 (May 16) 1941.

Of 1,638 children with appendicitis treated by the author between 1925 and 1938, 2.3 per cent died. For children 1 to 5 years of age the death rate was 7.8 per cent; for children 6 to 10 years of age, 2.5 per cent, and for children aged 11 to 15 years, 1.1 per cent. The mortality for boys was 3.27 per cent; for girls, 1.28 per cent. The high death rate in the youngest age group depends on diagnostic difficulties. Especially the initial symptoms and signs are often uncharacteristic, and, accordingly, the diagnosis is established too late and the child operated on too late. There are seldom any alarming symptoms at this age, and often the initial abdominal pains diminish after a while. Concomitant urinary symptoms or combination with enteritis increase the diagnostic failures.

WALLGREN, Göteborg, Sweden.

ULCUS DUODENI PERFORATUM IN A CHILD. ERIK THYSSEN, Ugesk. f. læger **103**:615 (May 8) 1941.

A boy of 11 years with uncharacteristic abdominal symptoms since 2 years of age was operated on with a tentative diagnosis of appendicitis. His condition grew worse, and he died seven days after the operation. Postmortem examination displayed a perforated duodenal ulcer.

WALLGREN, Göteborg, Sweden.

## Nervous Diseases

CONVULSIONS OF EARLY LIFE AND THEIR RELATIONSHIP TO CHRONIC CONVULSIVE DISORDERS AND MENTAL DEFECTS. DOUGLAS H. THOM, Am. J. Psychiat. **98**:574 (Jan.) 1942.

This paper represents the observation and experiences of the author as well as a study of the literature on the subject. In general summary, the author states:

"1. Convulsions repeated over a period of weeks or months during early life are more likely to be followed by epilepsy than a single convulsion or a series of seizures occurring in a short period (a few days or hours).

"2. A larger number have convulsions during the first year than at any other time. The peak in genuine epilepsy, according to one author, occurs around the second and the seventh to eighth year of life. The peak of symptomatic epilepsy is in the first year of life.

"3. Petit mal attacks during the first two years are likely to merge into chronic convulsions and become associated with mental retardation.

"4. The convulsion itself may cause permanent cerebral damage."

JAHR, Omaha.

WESTERN VARIETY OF EQUINE ENCEPHALITIS IN MAN: A CLINICOPATHOLOGIC STUDY. A. B. BAKER and H. H. NORAN, Arch. Neurol. & Psychiat. **47**:565 (April) 1942.

The authors report 10 additional cases of equine encephalitis, all the patients being adults 18 to 66 years of age. The onset is usually sudden, with headache, malaise, some nausea and vomiting and fever, followed in a few days by increased headaches, drowsiness which may progress to lethargy, and possibly vertigo. The spinal fluid cell count ranges from

normal to 200 cells per cubic millimeter. The only constant physical sign is mild stiffness of the neck associated with a positive Kernig sign. Positive diagnosis is made by neutralization tests. In 10 per cent of the cases the disease is fatal.

The disease has been reported in young children with sequelae of blindness and mental retardation, but no lasting sequelae have been reported in adults. BEVERLY, Chicago.

FACTORS AFFECTING CHANGES PRODUCED IN ELECTROENCEPHALOGRAM BY STANDARDIZED HYPERVENTILATION. H. DAVIS and W. McL. WALLACE, Arch. Neurol. & Psychiat. **47:** 606 (April) 1942.

Electroencephalographic tracings were made on 450 aircraft pilots after hyperventilation. By this method delta waves can sometimes be demonstrated, which are not present with normal breathing. BEVERLY, Chicago.

THE OLFACTORY-PAROTID REFLEX: STUDY OF ONE HUNDRED AND FIFTY PATIENTS WITH DISORDERS OF THE CENTRAL NERVOUS SYSTEM: A PRELIMINARY REPORT. C. A. ELSBERG, H. SPOTNITZ and E. I. STRONGIN, Arch. Neurol. & Psychiat. **47:**707 (May) 1942.

The olfactory-parotid reflex is the response of the glands to stimulation of the nasal mucosa with odorous substances. It is measured by the amount of secretion into an apparatus attached by suction over the opening of each Stenson duct. The resting rate of secretion was found to be abnormal in 56 per cent of a series of 64 patients with neurologic disorders.

BEVERLY, Chicago.

DIVERTICULA OF THE LATERAL VENTRICLES EXTENDING INTO THE CEREBELLAR FOSSA. A. E. CHILDE and F. L. MCNAUGHTON, Arch. Neurol. & Psychiat. **47:**768 (May) 1942.

The authors report a case of tumor in the brain of a 7½ year old girl in which there was protrusion of the lateral ventricle through the tentorium into the cerebellar fossa. These diverticula "should be diagnosed with a fair degree of certainty when one is aware of their occurrence" with the aid of ventriculograms. BEVERLY, Chicago.

INFLUENZAL MENINGITIS WITH RECOVERY. CASE REP., CHILDREN'S MEM. HOSP. **1:**17 (Feb.) 1942.

Current methods of therapy of influenzal meningitis are presented in conjunction with the report of a case of the disease in a 4-year old boy who recovered. Anti-influenzal rabbit serum was used in conjunction with sulfathiazole, sulfapyridine, sulfanilamide and sulfadiazine.

DUSHANE, Evanston, Ill.

ACUTE MENINGITIS. R. AMYOT, Union méd. du Canada **71:**140 (Feb.) 1942.

This is a well written résumé of the symptomatology, differential diagnosis, prognosis and treatment of the various forms of acute meningitis. DAFFINEE, Melrose, Mass.

A NOTE ON THE CEREBRAL CAUSES OF DEFECTS IN PERCEPTION AND CONSCIOUSNESS. F. PARKES WEBER, Brit. J. Child. Dis. **39:**15 (Jan.-March) 1942.

A labile vasomotor system may cause transitory emotional angiospastic attacks. Cerebral ischemia so caused may result in transient faintness or actual loss of consciousness. Angiospastic cortical blindness induced in flyers by evolutions, such as dive bombing, may have fatal results even if the attack does not progress to complete loss of consciousness.

One must bear in mind that transient symptoms may precede severe permanent ones.

LANGMANN, New York.

HYDROTHERAPY IN THE REHABILITATION OF NERVE LESIONS. MATTHEW B. RAY, Brit. J. Phys. Med. **5:**11 (Jan.) 1942.

The absence of a suitable pool is a serious handicap to recovery from the paralysis due to poliomyelitis. Splinting should be continued during pool treatment; otherwise the weight of a part may drag on affected muscles. Waterproof splints must be used.

Patients with spastic and ataxic types of cerebral paralysis are much improved by pool treatment. Patients with the myasthenic type gain more benefit from ordinary gymnastic exercises. Movement in water seems to defeat the purpose of treatment for patients with the athetoid type of paralysis. HARTMAN, Cleveland.

## Psychology and Psychiatry

RANGE OF PSYCHIATRIC MATERIAL WITHIN THE STATE SCHOOL. EDWARD J. HUMPHREYS and SUZANNE HOWE, Am. J. Psychiat. **98**:482 (Jan.) 1942.

This is a study of the range of material found within Letchworth Village in the state of New York. Its object was to illustrate the need for organized psychiatric treatment procedures. The case material of 32 patients is summarized from 150 case records of mental defectives and is divided into five major groups: (1) personality and character disorders associated with genetic and metabolic factors; (2) disorders following infection and injury; (3) disorders manifested in psychiatric and psychoneurotic reactions; (4) psychopathic states, and (5) behavior disorders of childhood.

Each one of these groups of disorders, the authors maintain, needs therapy different from that of the others. The oft-repeated statement "Once a defective, always a defective" needs modification.

JAHR, Omaha.

DELINQUENCY AND THE ELECTROENCEPHALOGRAPH. WARREN T. BROWN and CHARLES I. SOLOMON, Am. J. Psychiat. **98**: 499 (Jan.) 1942.

Seventeen per cent of 20 boys committed to a state training school for delinquents showed abnormal electroencephalographic records. Three of the records suggested petit mal; three showed irregularly abnormal patterns, and eleven showed a square-topped wave formation which the authors call "psychomotor-like." A few of these patients responded favorably to dilantin sodium.

JAHR, Omaha.

DEVELOPMENT AND CULTURAL PATTERNING IN CHILDREN'S PROTESTS. MARY SHIRLEY and LILLIAN POYNITZ, Child Development **12**:347 (Dec.) 1941.

Analysis based on verbatim quotations of the protests made by young children during the course of physical examinations revealed a developmental sequence for this type of speech. Successive stages in the development of protests beginning at 2 years are illustrated as follows: (1) nonadaptive cry, "Mam-ma"; (2) negative, "No! Don't!"; (3) alternative or excuse, "I want my clothes on"; (4) belligerence or threat, "I'm not going to get on the table; I'll cry"; (5) anticipated dread of specific technics, "I don't want a needle"; (6) modified protest, "Don't open my mouth *too* wide"; (7) nonadaptive and nonspecific, "Ouch." Girls tended to advance through this series of responses about six months in advance of boys; however girls more often appealed for comfort, whereas boys more often tended to express bravery. Such sex differences suggest the early impact of different cultural standards for boys and girls.

FROM THE AUTHORS' SUMMARY.

MOTOR CORRELATES OF INFANT CRYING. LOUISE BATES AMES, J. Genet. Psychol. **59**:239 (Dec.) 1941.

Since Darwin's book on the expression of the emotions in man and animals, relatively little study has been made of the crying behavior of infants. The present study conducted at the Yale Clinic of Child Development aims to determine if there are any specific motor patterns, other than the traditional face-mouth activity, associated with the crying of the infant.

Both crying and noncrying behavior were analyzed by means of photographic records. Five forms of behavior were found which commonly accompanied crying and which occurred infrequently in noncrying. They were: more activity than in noncrying; legs more active than arms; activity of legs more unilateral than bilateral; legs more flexed than extended, and foot to knee movements.

In general, noncrying behavior is characterized by less vigorous activity of limbs, extension of limbs, bilateral postures, greater activity of arms than legs, and holding of set postures.

The onset of crying was characterized by a somewhat standard pattern. Later activity followed no set chronologic or spatial pattern.

WEINFELD, Highland Park, Ill.

THE REACTIONS OF CHILDREN AND YOUTH TO WARTIME. BERT I. BEVERLY, J. Pediat. **20**: 665 (June) 1942.

The effects of war on the minds of children depends to a large extent on the attitudes of the parents. Good family morale exists when parents discuss possible risks and dangers with their children. Security and confidence in the parents are felt by the child. Children in England subjected to bombing who had security in their home and parents were better able to meet the

dangers than those who did not. For this reason evacuation of children from their homes is not to be advised.

To boost a child's morale further he should be given responsibility and a part to play, such as selling war stamps, or running errands for the Red Cross. Organizations such as the Y.M.C.A., the Boy Scouts and church and school groups play a large role in preventing moral deterioration.

STETTENBENZ, Buffalo.

PROPHYLACTIC ASPECT OF SCHIZOPHRENIA IN CHILDHOOD. J. LOUISE DESPERT, Nervous Child **1**:199, 1942.

The writer summarizes the available knowledge of the social and personal background of schizophrenia and the symptoms of the disorder. She stresses the lack of facilities and of adequately trained personnel as well as the need for further studies so that one may be able to recognize the syndrome before the onset of mental illness. Until this is done, specific prophylactic treatment is not possible.

BEVERLY, Chicago.

## Diseases of the Genitourinary Tract

NEPHROCALCINOSIS: A CASE REPORT. H. WOHL, Bull. Hosp. Joint Dis. **3**:105 (July) 1942.

A boy 11 years of age was brought into the children's urologic clinic because of nocturia and polydypsia. Excretion urograms showed extensive calcification in the parenchyma of both kidneys, which was thought to involve chiefly the medullary pyramids. There were also some ureteral dilatation and a dilated urinary bladder. The specific gravity of the urine was fixed at a low level; there were a heavy trace of albumin and a moderate number of leukocytes with an occasional erythrocyte. The phenolsulfonphthalein excretion was 45 per cent. Other tests showed normal conditions, except for a low serum albumin content, 2.4 per cent. Roentgenograms of the skeleton also showed nothing abnormal.

The giving of a citrate solution by mouth did not affect the renal calcification after a period of one month. The treatment at the time of writing consisted of 18,800 U. S. P. units of vitamin A and 3,400 U. S. P. units of vitamin D. The patient is being followed with the thought that there may develop skeletal changes similar to those of renal rickets.

LESLIE, Evanston.

SYMPOSIUM ON PYELONEPHRITIS: THE EFFECTIVENESS OF THE SULFONAMIDES ON THE BACTERIA ENCOUNTERED IN INFECTIONS OF THE UPPER PART OF THE URINARY TRACT. ROSCOE C. BORST, New York State J. Med. **42**:217 (Feb. 1) 1942.

The paper is one of a symposium on pyelonephritis presented at the annual meeting of the Medical Society of the State of New York.

No single drug has been discovered that is effective against all types of urologic bacterial infections. Sulfathiazole frequently becomes the drug of choice, especially against the more common organisms. Sulfadiazine is superior against streptococci and pneumococci, Friedländer bacilli and colon bacilli. Intravenous anesthesia should not be induced during therapy with any of the sulfonamide compounds. Other papers in the symposium were: "Chronic Pyelonephritis," by Drs. Reed M. Nesbit and Kyril B. Conger, and "Follow-Up Study of So-Called Pyelitis in Children," by Dr. Edgar Slotkin. Slotkin states that more children should receive urologic examinations, because of the high percentage of abnormalities that may be discovered.

AIKMAN, Rochester, N. Y.

PRECOCIOUS PUBERTY. R. BRIDGE and A. C. TELFER, M. J. Australia **1**:497 (April 25) 1942.

The authors report a case of precocious puberty in a boy aged 5 years whose muscular overdevelopment dated back to early infancy. No cause for the condition could be found. Adrenal tumor was excluded by exposure of both glands.

GONCE JR., Madison, Wis.

SEMINOMA OF THE TESTICLE IN AN INFANT. ROBERTO VALDÉS DÍAZ, SANCHEZ SANTIAGO, DÍAZ ROUSSELOT and CARBONELL SALAZAR, Arch. de med. inf. **9**:23 (Jan.-March) 1942.

An 8 month child had seminoma of the testicle which was treated radiologically. Recovery apparently took place, and the child had remained without further symptoms.

SANFORD, Chicago.

URETERAL FISSURES. J. M. HERRERA, Rev. méd. latino-am. **27**:653 (April) 1942.

The literature on ureteral anomalies reported during the period between 1900 and 1938 is reviewed. The study, comprising 110 cases, reveals that the anomaly occurs nearly three times as frequently in the female as in the male (72 to 28 per cent) and on the right side more frequently than the left (52 to 41 per cent). The author discusses the embryologic considerations and the classification. Line drawings illustrate the more frequent types.

DWYER, Kansas City, Mo.

## Diseases of the Ductless Glands; Endocrinology

ON THE MECHANISM OF ENHANCED DIABETES WITH INFLAMMATION. VALY MENKIN, Am. J. Physiol. **134**:517 (Oct.) 1941.

An acute pleural inflammation in a dog fails to alter its blood sugar level. On the other hand, the presence of an acute inflammatory reaction in a dog rendered diabetic by pancreatectomy induces a rapid and pronounced elevation in the blood sugar level.

The extent of local proteolysis at the site of an acute inflammation in a diabetic dog is considerably more pronounced than that found in a nondiabetic animal. This is indicated by a lower concentration of the total proteins and correspondingly higher concentrations of urea, nonprotein nitrogen and amino acid nitrogen than are encountered in the exudative material of a nondiabetic animal.

The enhanced protein metabolism in the inflamed area of a diabetic animal is correlated with a sharp elevation in both exudative sugar and exudative lactic acid. Administration of insulin reduces not only the level of sugar and lactic acid in such diabetic exudates, but this substance diminishes as well the degree of local proteolysis. This fact supports the view that gluconeogenesis at the site of an acute inflammation in a diabetic animal originates from protein through deamination of the molecule.

Enhanced local proteolysis in the inflamed area of a diabetic animal implies increased damage to the tissue. The cytologic picture of a diabetic exudate indicates that polymorphonuclear leukocytes manifest pronounced signs of cellular injury when compared with similar cells derived from a normal exudate.

The biochemical changes encountered in the exudate of a depancreatized dog are similarly reflected in its circulating blood. Besides an elevation in blood sugar level, there is also an increase in the blood concentration of nonprotein nitrogen, urea and amino acid nitrogen. Studies after pancreatectomy or splenectomy and a comparison of the relative dextrose concentrations in exudate and in blood indicate that the raised levels of carbohydrate and nitrogenous constituents of the systemic circulation are due neither to the diabetes per se nor to the operative procedure. The elevation in the levels of these substances is referable to an absorption from the area of acute inflammation which complicates the diabetic disorder. Insulin, by inhibiting formation of dextrose in the inflamed area, likewise prevents a rise in the blood stream of intermediary products of carbohydrate and protein metabolism.

The available evidences, therefore, support the view that the mechanism of enhanced diabetes with concomitant inflammation might well be referable primarily to an increased local proteolysis in the inflamed area, favoring a combined picture of increased tissue damage with a corresponding elevation in dextrose formation; the dextrose, in turn, gradually diffuses into the systemic circulation.

Macpherson and co-workers have shown that the administration of a vitamin K analogue, either to the mother between twelve and four hours before delivery or to the infant within twenty-four hours of birth, maintains the prothrombin index at a higher and protective level during the first few days of life. In any case of manifest or suspected hemorrhage in a newborn infant, vitamin K should be administered at once if it has not been given prophylactically. . .

NOURSE, Cleveland.

RESPONSE TO GROWTH HORMONE OF HYPOPHYSECTOMIZED RATS WHEN RESTRICTED TO FOOD INTAKE OF CONTROLS. WALTER MARX, MIRIAM E. SIMPSON, WILLIAM O. REINHOLDT and HERBERT M. EVANS, Am. J. Physiol. **135**:614 (Feb.) 1942.

When care was taken to secure an identical intake of food, hypophysectomized rats treated with a purified preparation of the growth factor from the anterior lobe of the pituitary gland gained significantly more weight than the untreated controls. This would indicate that the growth factor caused increased deposition of tissue substance, not as a consequence of increased intake of food but through better utilization of the consumed food. All internal organs

examined were heavier in the groups treated with growth preparation. They grew only approximately at the same rate as did the body as a whole, with the exception of the thymus, which grew considerably faster.

NOURSE, Cleveland.

EFFECT OF THYROID MEDICATION ON BRAIN METABOLISM OF CRETINS. H. E. HIMWICH, C. DALY and J. F. FOGERAS, Am. J. Psychiat. **98**:489 (Jan.) 1942.

Eleven cretins ranging in age from 9 to 31 years were studied. After the patients had been without thyroid medication, two months of repeated determinations of basal metabolism and oxygen studies on the blood from the internal jugular vein were made. Thyroid was then administered. An average increase of 32 per cent in cerebral metabolism was revealed as a result of administration of thyroid.

JAHR, Omaha.

ELECTROENCEPHALOGRAPHIC STUDIES AND DELINQUENT BEHAVIOR PROBLEM CHILD. NORMAN Q. BRILL, HERTA SEIDEMANN, HELEN MONTAGUE and BEN BALZER, Am. J. Psychiat. **98**:494 (Jan.) 1942.

This study represents a report on 28 children who had been brought before the Court of Domestic Relations in the city of New York. Sixty-one per cent of the entire group had electroencephalographic potentials suggestive of dysfunction of the brain.

JAHR, Omaha.

THE GONAD-STIMULATING POTENCY OF THE PITUITARY OF HYPOTHYROID YOUNG MALE RATS. KATHRYN F. STEIN and MARGARET LISLE, Endocrinology **30**:16 (Jan.) 1942.

The data indicate a decrease in gonad-stimulating potency of the pituitary gland in young male rats twenty-one to fifty days after thyroidectomy.

JACOBSEN, Buffalo.

DOES A STIMULATION OF THE GERMINATIVE EPITHELIUM OF IMMATURE MALE RATS OCCUR AFTER TREATMENT WITH GONADOTROPIC HORMONES, AUGMENTATIVE SUBSTANCE, ANDROGENS AND VITAMIN E? BERNHARD ZONDEK, ARON BRZEZINSKI and FELIX SULMAN, Endocrinology **30**:25 (Jan.) 1942.

None of the substances used brought any acceleration of spermatogenesis.

These experiments support the supposition that spermatogenesis in mammals is a complex process, in which, besides the hormones so far studied, several still unknown factors are involved. Precocious maturation of the ovaries is a relatively simple process, involving the use of gonadotropin only.

JACOBSEN, Buffalo.

MAMMARY GLAND DEVELOPMENT WITH MAMMOGEN I IN THE CASTRATE AND THE HYPOPHYSECTOMIZED RAT. A. A. LEWIS, E. T. GOMEZ and C. W. TURNER, Endocrinology **30**: 37 (Jan.) 1942.

These data show that lipid anterior pituitary extracts will cause growth of the mammary duct systems of castrate male and hypophysectomized male and female rats.

JACOBSEN, Buffalo.

STUDIES ON RECOVERY OF THE REPRODUCTIVE SYSTEM OF THE MALE RAT FROM REGRESSIVE CHANGES INDUCED BY STILBESTROL. CHARLES S. MATTHEWS, EDWARD L. SCHWAGE and FREDERICK E. EMERY, Endocrinology **30**:89 (Jan.) 1942.

Male rats treated with diethylstilbestrol became sterile, but 13 of 17 recovered in forty-seven days sufficiently to effect successful impregnation. The male organs of reproduction have the capacity for gaining rapidly in weight and regenerating new tissue cells after undergoing regressive changes induced by diethylstilbestrol.

JACOBSEN, Buffalo.

EFFECTS OF TESTOSTERONE PROPIONATE IN IMMATURE AND ADULT FEMALE RATS. G. L. LAQUEUR and C. F. FLUHMANN, Endocrinology **30**:93 (Jan.) 1942.

As a result of the experiments reported the theory is advanced that testosterone propionate induces in female rats a functional transformation of the anterior lobe of the hypophysis characterized by an inhibition of the so-called luteinizing or interstitial cell-stimulating hormone and an increased production of the follicle-stimulating hormone.

JACOBSEN, Buffalo.

THE PREPARATION AND PROPERTIES OF UROGASTRONE. JOHN S. GRAY, E. WIECZOROWSKI, J. A. WELLS and S. C. HARRIS, Endocrinology **30**:129 (Jan.) 1942.

Urogastrone is a water-soluble substance obtainable from the urine of both dogs and human beings which effectively inhibits gastric secretion. Methods are described for the preparation of extracts containing urogastrone from normal urine. From its chemical behavior the active agent appears to be neither a protein nor a polypeptide susceptible to the action of pepsin but is probably a complex organic base. Its various effects are recorded.

JACOBSEN, Buffalo.

CAPACITY OF RAT'S LIVER TO INACTIVATE DESOXYCORTICOSTERONE ACETATE. M. W. BURRILL and R. R. GREENE, Endocrinology **30**:142 (Jan.) 1942.

Burrill and Greene conclude that the liver can inactivate desoxycorticosterone acetate but that this ability has quantitative limitations.

JACOBSEN, Buffalo.

ELIMINATION OF THE PARS NERVOSA WITHOUT ELICITING DIABETES INSIPIDUS. ALLEN D. KELLER, Endocrinology **30**:408 (March) 1942.

When the junctional region between the hypophysis and the hypothalamus was undisturbed, hypophysectomy did not produce diabetes insipidus in dogs and cats. Infringement of the anterior portion of the hypothalamus invariably produced diabetes insipidus. The existence of an extra hypophysial antidiuretic-elaborating mechanism is suggested. JACOBSEN, Buffalo.

INFLUENCE OF TESTOSTERONE ON THE MORPHOGENETIC ACTIONS OF ESTRADIOL. S. ALBERT, Endocrinology **30**:454 (March) 1942.

Albert shows that many of the observable effects of the administration of estradiol to male rats are inhibited by the simultaneous administration of testosterone. JACOBSEN, Buffalo.

SEX HORMONES AND HAIR CHANGES IN RATS. THOMAS R. FORBES, Endocrinology **30**:465 (March) 1942.

Subcutaneous implantation of pellets of various sex hormones in crystalline form was usually followed by partial pigmentation of the fur of albino rats and by alopecia in brown rats.

JACOBSEN, Buffalo.

THE INFLUENCE OF STILBESTROL AND THYROXIN ON GALACTOSE ABSORPTION AND LIVER FUNCTION. ROBERT C. GRAUER, WILLIAM F. STARKEY and ELEANOR SAIER, Endocrinology **30**:474 (March) 1942.

Injections of thyroxin in dogs increased the rate of absorption of galactose from the intestinal tract and eventually produced damage to the liver, resulting in increased galactose in the blood.

Diethylstilbestrol increased the rate of absorption and hastened the clearance from the blood.

JACOBSEN, Buffalo.

TOXICOLOGY OF THE SYNTHETIC ESTROGEN DIETHYLSTILBESTROL AND CERTAIN RELATED COMPOUNDS. R. S. TEAGUE, J. Pharmacol. & Exper. Therap. **75**:145 (June) 1942.

The studies were made on male and female rats given large daily doses of a synthetic estrogenic substance (diethelystilbestrol, diethylstilbestrol dipropionate, diethylstilbestrol monomethyl ether, diethylstilbestrol monomethyl ether acetate, diethylhexestrol, diethylhexestrol monomethyl ether and estradiol were the substances investigated) for from ten to eighty-six days. No animals died, but all products depressed growth. A characteristic change was enlargement of the liver due to excessive deposit of glycogen. The adrenals showed certain changes probably related to vascular injury after the larger doses. Other organs, kidneys, ovaries, uterus and testes, showed changes probably not characteristic.

PILCHER, Cleveland.

"GROWTH" AND THE DIABETOGENIC ACTION OF ANTERIOR PITUITARY PREPARATIONS. F. G. YOUNG, Brit. M. J. **2**:897 (Dec. 27) 1941.

This article is an interesting, complicated and detailed report of experiments done on puppies and adult dogs into which pituitary extracts had been injected. The article does not lend itself to abstracting, as the discussion and reasoning of the various points made are

rather long and detailed. However, the author nicely summarizes the work done, and the following statements are included in this report.

"When puppies are treated daily with doses of crude pituitary extract greatly in excess of those required to produce diabetes in adult dogs they respond with an increased growth rate but do not exhibit symptoms of diabetes. When one puppy was treated daily for nearly five months it eventually became intensely diabetic, and then stopped growing despite continuation of treatment.

"When adult dogs that have been rendered permanently diabetic by a short period of pituitary treatment are given a single injection of pancreotropic pituitary extracts, which produces no detectable diabetogenic activity in normal adult dogs, the response may be a transitory attenuation of the diabetic condition with nitrogen retention and increase of body weight or an exacerbation of the diabetes with loss of nitrogen and of body weight; intermediate responses were also obtained. These differences in response are believed to result largely from differences in functional capacity of pancreatic islet tissue.

"It is suggested that an increase in body weight, associated with an increase in function of the pancreatic islets, can be regarded as neutralizing, for a time at least, the diabetogenic influence of the anterior pituitary gland. It is therefore possible that the excessive weight of the adult diabetic patient and the abnormal height of the diabetic child (White) indicate an increased pituitary function which may be of temporary duration only, and of which the diabetogenic action may be completely masked for a time by a protective increase in pancreatic islet activity, induced by the pancreotropic action of the gland. Such a condition might lead first to an increase in the mass of the body and eventually to diabetes."

ROYSTER, University, Va.

THE GALACTOSE TOLERANCE TEST IN THYREOTOXICOSIS. T. E. WILSON, M. J. Australia **1**:33 (Jan. 10) 1942.

Wilson's studies indicate that the elevation in the blood galactose tolerance curve in thyrotoxicosis is chiefly due to accelerated intestinal absorption and to a lesser extent to impairment of the glycogenic function of the liver. The literature dealing with carbohydrate metabolism in thyrotoxicosis is reviewed.

GONCE JR., Madison, Wis.

DIABETIC DWARFISM WITH HEPATOMEGALY. W. G. OAKLEY, Proc. Roy. Soc. Med. **35**:540 (June) 1942.

The patient, a boy of 7½ years, was taken sick in 1939 with diabetes. He was treated by diet and given 6 units of insulin twice a day. Later he was admitted to the hospital, where the insulin was increased to 10 units twice a day (soluble insulin). Two and a half years later he was found to have the characteristic appearance, with "moon" face, infantile hands and enlarged liver. The blood sugar level was found to be fluctuating greatly. The treatment was changed to 12 units of protamine zinc insulin with 8 units of soluble insulin once a day, in the morning before breakfast.

WILLIAMSON, New Orleans.

SYNDROMES OF DEVELOPMENTAL DISTURBANCES. LUIS V. CUERVO, Rev. méd. cubana **53**:461 (June) 1942.

The most frequent of these syndromes are macrosomia, microsomia, backwardness of motor or of intellectual development, disturbances in dentition and in closure of fontanels, infantilism, gigantism, premature union or delay of union of diaphysis and epiphysis, disturbances of the osteoligamentomuscular apparatus, precocious puberty, adiposogenital syndrome, feminoid or viroid manifestations, eunuchism, gynecomastia, microgenitosomia, bashfulness, insipid polyuria, hypocalcemia, goiter of puberty, overweight, overheight and disturbances of general capacity.

SANFORD, Chicago.

CASE OF LAURENCE-MOON-BIEDL SYNDROME. ERIK MOGENSEN, Nord. med. (Hospitalstid.) **10**:1437 (May 10) 1941.

A boy of 7 years with slight diabetes insipidus, deformity of the skull, brachydactylia and pes varus showed the typical signs of the Laurence-Moon-Biedl syndrome: adiposogenital dystrophy, retinitis pigmentosa, mental deficiency and polydactylia. The parents and two brothers were healthy; a younger sister suffered from hydrocephalus. Treatment with a gonadotropic substance resulted in an untoward sexual development with slight signs of precocious puberty. Treatment with thyroid reduced the adiposity. No real improvement of the condition could be attained by this endocrine treatment.

WALLGREN, Göteborg, Sweden.

OBSERVATIONS ON HORMONE TREATMENT OF HYPOGALACTIA. C. GYLLENSWÄRD, Nord. med. (Hygiea) **11**:2708 (Sept. 20) 1941.

Galactagogic substances from the hypophysis and from the corpus luteum (prolactin and progestin) were tried without effect in 3 cases. The author stresses the importance of strict technic and of psychic factors in the treatment of hypogalactia.

WALLGREN, Göteborg, Sweden.

## Diseases of the Muscles, Bones, Joints and Lymph Glands

TIME OF APPEARANCE AND FUSION OF OSSIFICATION CENTERS AS OBSERVED BY ROENTGENOGRAPHIC METHODS. H. FLECKER, Am. J. Roentgenol. **47**:97 (Jan.) 1942.

The author finds that no comprehensive survey of the time of appearance and fusion of ossification centers by use of roentgenographic means has yet been undertaken. Study of ossification time was made on human fetuses from 30 mm. to full term, as well as on the skeleton from birth to maturity. This study, however, is not complete. The work includes individual study of the bones of the hand and foot, including the sesamoids. Variations from the normal, such as inconsistency of appearance, asymmetry, sexual and racial differences, are included.

The importance of a thorough study of the normal development is stressed in relation to further evaluation of the effects of endocrine disturbances and other pathologic states.

A chronologic table of appearance and a summarized table of findings are included in the work.

SQUIRE, Chicago.

MALIGNANT TUMORS DEVELOPING IN SACROCOCCYGEAL TERATOMATA. HERMAN LISCO, Ann. Surg. **115**:378 (March) 1942.

The author reports 2 cases of typical congenital sacrococcygeal teratoma in children. In both cases the growth was found at or soon after birth, and in each of them a papillary adenocarcinoma with widespread metastases developed.

Ten similar cases reported in the literature are reviewed.

Because of the possibility of malignant degeneration in sacrococcygeal teratomas, the necessity of early surgical removal of these growths is emphasized.

BANTIN, Omaha.

MYASTHENIA GRAVIS TREATED BY EXCISION OF THYMIC TUMOR: REPORT OF TWO CASES. ELDRIDGE CAMPBELL, NATHAN F. FRADKIN and BASILE LIPETZ, Arch. Neurol. & Psychiat. **47**:645 (April) 1942.

The authors add another to the series of cases of myasthenia gravis in which improvement has been reported after removal of the thymus gland. This is further evidence that benign tumor and hyperplasia of the adult thymus are in some way associated with myasthenia gravis.

BEVERLY, Chicago.

OBSERVATIONS ON THE NATURE OF MYASTHENIA GRAVIS: THE PHENOMENA OF FACILITATION AND DEPRESSION OF NEUROMUSCULAR TRANSMISSION. A. M. HARVEY, J. L. LILIENTHAL JR. and S. A. TALBOT, Bull. Johns Hopkins Hosp. **69**:547 (Dec.) 1941.

1. The abnormality of neuromuscular transmission in the hands has been studied in 5 patients with myasthenia gravis.

2. In 3 patients it was found that a single maximal nerve stimulus was capable of exciting only a portion of the muscle innervated.

3. In 1 patient facilitation in transmission similar to that described by Bremer and Homès and by Brown in partially curarized animals was observed. A train of four nerve volleys at intervals of 16 and 32 milliseconds resulted in action potentials of increasing magnitude until a normal value was reached. The voltage of the response to a single stimulus was raised to a similar value after the administration of prostigmine.

4. In another patient the depression following the passage of a single nerve volley was so severe that transmission could not be restored to normal by prostigmine.

5. In 2 other patients with myasthenia gravis with no clinical involvement of the hands, the curves for two volley transmission were normal but a latent myasthenic defect of transmission was revealed by the depression in a rapid train of responses.

6. The degree of this depression apparently parallels the severity of the myasthenia in the muscle under study.

7. The relation of these observations to the pathogenesis of myasthenia gravis is discussed.

FROM THE AUTHORS' SUMMARY.

OBSERVATIONS ON THE NATURE OF MYASTHENIA GRAVIS: THE INTRA-ARTERIAL INJECTION OF ACETYLCHOLINE, PROSTIGMINE AND ADRENALINE. A. M. HARVEY and J. L. LILIENTHAL JR., Bull. Johns Hopkins Hosp. **69**:566 (Dec.) 1941.

Prostigmine produces local and then general return of motor power. It fails to evoke the local muscle fasciculations and does not produce the motor weakness observed in normal subjects.

Acetylcholine, in addition to producing local pain, flushing and sweating, evokes a powerful, sustained contraction of the inoculated muscles. The latter effect is in sharp contrast to the transitory weakness evoked in normal subjects.

Epinephrine produced a moderate return of local motor power in a myasthenic patient with weakness of the hands.

These observations and related physiologic data indicate that the fundamental defect in myasthenia gravis may be a reduction in the normal quantity of proper transmitting agent released, or available for release, by the terminal branches of the motor nerves at the neuromuscular junction. The reduction in the quantity of transmitter released may be masked to a variable degree by an increased sensitivity of the end plate of the muscle to the transmitting agent.

The analogy between myasthenia gravis and partial curarization suggests the hypothesis that a circulating inhibitor substance may be responsible for the primary neuromuscular defect. There is evidence to suggest that this hypothetic substance may arise from the thymus.

FROM THE AUTHORS' SUMMARY.

CONGENITAL LACUNAR SKULL ("LÜCKENSCHÄDEL"). H. F. BETTINGER, M. J. Australia **1**:526 (May 2) 1942.

The history of lacunar skull is briefly reviewed. The mechanism involved in the development of the condition may be summarized as follows: Toward the end of pregnancy occurs a physiologic incongruity between skull and brain. Under normal conditions localized effects are not possible, because the cerebrospinal fluid, acting like a water cushion, insures an equal pressure on all parts of the cranium. In meningocele, however, the cerebrospinal fluid escapes into the sac and the brain thus comes into direct contact with the inner surface of the vault. Additional space for the growing brain cannot be obtained by bulging of the nonosseous parts of the cranium, because the amniotic fluid around the skull obviates all differences of pressure. Erosion of the bone by pressure atrophy will therefore occur wherever the gyri press most closely against the inner table. A typical lacunar skull will thus develop.

GONCE JR., Madison, Wis.

## Diseases of the Eye

THE EARLY DIAGNOSIS OF CHOROIDAL MELANOMAS. B. RONES, Am. J. Ophth. **25**:39 (Jan.) 1942.

Rones discusses the diagnosis of choroidal melanomas and emphasizes their high mortality rate. He cites 2 cases which illustrate how important it is to make an early diagnosis and to remove all suspicious blind eyes even though occasional mistakes be made.

W. S. REESE [ARCH. OPHTH.].

FAMILIAL INCIDENCE OF RETINOBLASTOMA. H. F. FALLS, Am. J. Ophth. **25**:42 (Jan.) 1942.

Falls reports on a family in which retinoblastoma developed in 6 of a total of 10 siblings. Two children with monocular involvement survived. Of the 4 who died, 3 displayed bilateral involvement. Falls strongly urges sterilization of parents who have produced 2 children with retinoblastoma.

W. S. REESE [ARCH. OPHTH.].

SULFATHIAZOLE IN THE TREATMENT OF GONORRHEAL EYE DISEASE. C. R. MULLEN, Am. J. Ophth. **25**:59 (Jan.) 1942.

Mullen found that sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) used locally and internally in 5 cases of gonorrheal disease of the eye gave as satisfactory results as those obtained from sulfanilamide used similarly. He stresses the importance of frequent irrigations.

W. S. REESE [ARCH. OPHTH.].

TREATMENT OF RETINOBLASTOMA (RETINAL GLIOMA) SURGICALLY AND BY IRRADIATION. HAYES MARTIN and ALGERNON B. REESE, Arch. Ophth. **27**:40 (Jan.) 1942.

This is an exhaustive report of 23 cases of retinoblastoma (glioma), with special reference to the treatment of bilateral glioma by enucleation of the more involved eye and irradiation of the other in an attempt to save life, control the disease and preserve vision. Considering the seriousness of the situation the results are remarkable. The selection of the method of treatment and the technics of the various types of treatment are well described.

Of the 10 patients having bilateral glioma treated by having one eye enucleated and the other irradiated, 8 were living and well, 5 with vision and 3 without vision, and 2 were dead. Of the 7 patients with residual and recurrent retinoblastoma, 3 were living and well.

WESCOTT, Chicago.

PIGMENTARY DEGENERATION OF THE RETINA. P. R. McDONALD and F. H. ADLER, Arch. Ophth. **27**:264 (Feb.) 1942.

Accepting the fact that retinitis pigmentosa is a hereditary abiotrophic defect should not prohibit an attempt to discover factors in its causation. The authors are here concerned with the part that the melanophore hormone, secreted by the pars intermedia of the pituitary gland, may play in the pigment layer of the retina. They came to the following conclusions:

"1. The injection of purified melanophore hormone has no effect on retinal pigment migration in the frog.

"2. The injection of melanophore hormone has no effect on the retinal pigment of the rat.

"3. Hypophysectomy, complete or incomplete, has no effect on retinal pigment migration in the frog.

"4. The assay for an active melanophore-expanding substance in the blood of patients with retinitis pigmentosa is not of significant diagnostic value."

WESCOTT, Chicago.

CHRONIC CHOROIDITIS PRODUCED WITH STREPTOCOCCUS VIRIDANS AND STREPTOCOCCUS HAEMOLYTICUS IN NORMAL AND IN IMMUNIZED RABBITS. SIDNEY ROTHBARD and D. MURRAY ANGEVINE, J. Infect. Dis. **70**:201 (May-June) 1942.

Chronic choroiditis was produced in two thirds of the normal rabbits that received multiple intravenous injections of a broth culture (4 cc.) of Streptococcus viridans. Single injections of the culture and repeated injections of suspensions of killed bacteria or of filtrates or of carbohydrate or protein fractions of Str. viridans did not produce this lesion.

Chronic choroiditis appeared in all normal rabbits that received repeated courses of daily intravenous injections of a broth culture (1 to 2 cc.) of Str. viridans alternated with periods of rest.

In approximately half of the rabbits intravenously immunized with killed cultures of Str. viridans choroiditis developed after intravenous injections of small doses of a homologous culture, whereas control animals with no immunization did not manifest uveal inflammation.

A large proportion of rabbits (43 per cent) intravenously immunized with killed cultures of Str. haemolyticus and almost as large a proportion of those intracutaneously immunized (39 per cent) acquired choroiditis after intravenous injections of homologous cultures, whereas of rabbits with no immunization few (4 per cent) acquired choroiditis under the same conditions. After intravenous or intracutaneous immunization choroiditis was often caused by a single intravenous injection.

These experiments demonstrate that with the quantities of culture used, previous intravenous or intracutaneous immunization increased the susceptibility of rabbits to the acquirement of chronic inflammation of the choroid.

TOOMEY, Cleveland.

PRIMARY OCULAR TUBERCULOSIS. P. J. NORDENFELT, Nord. med. (Hygiea) **11**:2719 (Sept. 20) 1941.

A boy of 13 years fell ill with high fever and swelling of the left eyelid and of the left preauricular region. At the examination, thirteen days later, he still had fever and an increased sedimentation rate, and there was voluminous lymphadenitis at the left preauricular region and at the angulus mandibulae. There was an ulceration culture of which produced a positive reaction in a guinea pig at the left upper eyelid with scanty tubercles in the neighborhood. A tuberculin test gave a positive reaction. The boy was still under treatment with Finsen rays. The source of infection was not detected.

WALLGREN, Göteborg, Sweden.

## Skin Diseases; Allergy

PRURITUS IN MEDICINE AND IN DERMATOLOGY. MAX S. WIEN, Illinois M. J. **81**:125 (Feb.) 1942.

Wien presents a discussion of cutaneous entities which need be considered in cases of pruritus, enumerates signal points in differential diagnosis and comments on therapy. A patient with pruritus who presents cutaneous signs may be suffering from a cutaneous entity of exogenous origin or of varied internal origin. Pruritus may be cutaneous expression of systemic disease.

BARBOUR, Peoria, Ill.

DERMATITIS DUE TO DEXTRINS USED AS AN ADHESIVE ON TAX STAMPS. J. D. WALTERS and E. C. STERN, J. A. M. A. **116**:1518 (April 5) 1941.

Walters and Stern report 2 instances of cutaneous sensitivity to a dextrin in female workers who were employed to affix tax stamps and who thus came into prolonged contact with the material in solution. The dextrin is a component of the adhesive surface of tax stamps, as well as of United States postage stamps.

LEWIS, New York. [ARCH. DERMAT. & SYPH.]

POTASSIUM CHLORIDE THERAPY AND SERUM POTASSIUM IN INFANTILE ECZEMA. A. V. STOESSER, Proc. Soc. Exper. Biol. & Med. **49**:332 (March) 1942.

Six infants with eczema ranging in age from 7 to 16 months were chosen and placed under constant environmental conditions in the hospital. The type of eczema was practically the same in each. There was a moist, red surface exuding serum. Three of the patients received potassium chloride orally, the dose starting with 5 grains (0.32 Gm.) three times daily and increasing gradually over a period of one week to 15 grains (0.97 Gm.) four times a day. The remaining 3 infants were observed as controls.

Samples of blood for the determination of serum potassium, sodium and chloride were obtained shortly after admission to the hospital during acute stages of the disease, two weeks later and again four weeks later.

The ingestion of potassium chloride does not improve the eczema but instead causes an aggravation of the cutaneous condition. The serum potassium is above the normal range during the more severe phases of the disease. This elevation is present in infants receiving the potassium chloride as well as in the controls. With improvement in the eczema, the potassium level of the blood begins to fall; when all the oozing has disappeared and the skin is dry, the values are normal.

HANSEN, Minneapolis.

ALLERGY, ENURESIS AND STAMMERING. I. GORDON, Brit. M. J. **1**:357 (March 14) 1942.

This article is an investigation as to the incidence of enuresis and stammering in patients with allergic diseases. Of 128 bed wetters it was found that 65 per cent either had a history of allergy in the family or had allergy themselves. In a control series the figure was only 28 per cent. Of the various classes of allergy, migraine was by far the most common, with a 48 per cent incidence, asthma, hay fever and eczema being less frequent in that order. Because of the high incidence of a history of migraine found in patients with enuresis, the incidence of headaches was then obtained in the children with enuresis. In the control group only 13 per cent of 200 normal patients complained of headache, while of 50 bed wetters 44 per cent complained of severe headaches.

In the discussion the author points out that it has long been established that persons with asthma suffer from somewhat abnormal mental states, usually of a minor and vague type. It is also noted that asthmatic attacks are often brought on by some psychologic stimulus. Because of the universal use of a headache as an excuse to retire from some embarrassing or unfavorable situation, it is pointed out that it is not surprising that headaches are found frequently in bed-wetting patients. In his summary the author makes the following statements:

"It is confirmed that enuresis and stammering are commoner among allergics.

"It is suggested that among the manifestations of allergy migraine is largely associated with these two complaints, and in fact that headaches of various types may be a factor in the aetiology.

"This being the case, as well as the fact that nightmares are so common in allergic children, it is suggested that enuresis and stammering are not themselves direct allergic manifestations but result from the abnormal psychological traits of allergics."

BIRDSONG, University, Va.

A New Method of Controlling the Head Louse. J. R. Busvine and P. A. Buxton, Brit. M. J. **1**:464 (April 11) 1942.

Attention is called to the extremely high incidence of head lice among women in England. It is stated that 30 to 50 per cent of young women in industrial areas in Britain have head lice. Reference is made to the old methods of combating the parasites, and it is stated that the usual methods are not too successful and are rather objectionable to the patient. The improved method suggested in this article is the use of one of three new medications: (1) a 25 per cent solution of lauryl thiocyanate dodecyl thiocyanate in liquid petrolatum; (2) 50 per cent lethane (12.5 per cent N-butyl carbitol thiocyanate); 37.5 betathiocyanoethyl laurate) in a similar oil, and (3) derris cream (1 per cent rotenone and 7 per cent derris extract in lanette wax). Detailed instructions as to how these creams are made up may be found in the article. The advantage of the method is that one good application should produce a cure. After the application of the medication the head is not washed for a period of ten days. The number of failures following use of one of these three methods was much lower than with the accepted methods now used. The objection to this treatment was that there is occasionally some irritation of the scalp; because the medicaments occasionally cause irritations on other parts of the body, they should not be used except on the scalp.

That the head should not be washed for seven to ten days is one of the objections usually raised. The advantage of this method is that it gives a greater percentage of cures and produces a less objectionable odor and appearance.

Birdsong, University, Va.

Universal Calcinosis in an Infant. B. Sanchez Santiago and Raul Pereiras, Arch. de med. inf. **9**:1 (Jan.-March) 1942.

Roentgenograms are shown of a 14 month old child in whom symptoms of calcinosis began at 5 months. These were localized edema and swellings of the extremities. Later these became plaques, which biopsy and roentgenograms showed to be calcium.

Sanford, Chicago.

## Teeth and Dentistry

Local Implantation of Sulfanilamide and Its Derivatives in Wounds. Samuel P. Harrison and J. Albert Key, Arch. Surg. **44**:22 (Jan.) 1942.

The authors attempted to determine whether the employment of sulfanilamide and its various derivatives in wounds inhibits the process of healing or leads to the formation of adhesions in the peritoneum. Adult guinea pigs and white rats were utilized in this study. It was concluded that the local implantation of a moderate amount of sulfanilamide powder in wounds of the abdominal wall and in wounds of the stomachs of the experimental animals does not appreciably interfere with the healing of these wounds. Such wounds when tested showed the same tensile strength as similar wounds in which sulfanilamide was not implanted. Also it was noted that the introduction of sulfanilamide, sulfathiazole or sulfadiazine into the peritoneal cavity does not tend to cause adhesions in rats.

Kaiser, Rochester, N. Y.

## Miscellaneous

Ollier's Disease: Report of the First Case with Involvement of the Optic Nerve. Joseph L. Nielson Jr., Bull. Los Angeles Neurol. Soc. **6**:104 (Sept.) 1941.

Nielson reports the case of a girl aged 17 years who had begun at the age of 3 to exhibit progressive shortening of the left leg. When she was 12 years old pain in the left shoulder was followed by inability to raise the left arm higher than the shoulder. Four months before her examination diplopia appeared, resulting from progressive ophthalmoplegia with ptosis, which soon became complete. On examination there were primary atrophy of the optic nerve with great reduction of vision, nearly complete ptosis and both external and internal ophthalmoplegia on the right side. Sensation was impaired over the distribution of the first two divisions of the right trigeminal nerve, and there were weakness and atrophy of the right temporal and masseter muscles. Roentgenographic study revealed erosion of the right optic foramen. Areas of decreased density, coarsened trabeculation and small regions of calcification in otherwise clear zones were found in the ends of long bones, the scapula, the ilium and many other bones. The levels of calcium and phosphorus in the serum were normal.

Nielsen identifies the condition with the dyschondroplasia of Ollier because of its non-familial incidence in early childhood, the shortening of the limb, the normal levels of calcium

and phosphorus in the blood and the characteristic roentgenologic features. The last-mentioned changes consist of rarefactive areas in the ends of the long bones on the diaphysial side of the epiphysial line, with striping due to septums 0.5 to 1.0 cm. apart lying in the long axis of the bones. Smaller bones may also be involved. The radiopaque areas are found on biopsy to consist of dysplasic cartilage. Nielson states that his case is unique in that implication of the optic nerve has not been reported previously. The treatment is symptomatic, and the course tends to gradual improvement.

MACKAY, Chicago. [ARCH. NEUROL. & PSYCHIAT.]

THE CENTENNIAL OF THE GOLDEN AGE OF ANESTHESIA. JOSEPH A. DOHERTY, J. M. A. Georgia **31**:106 (March) 1942.

This is an editorial which appeared in "The Apollonian" (Boston) for January 1942. It extols Dr. Crawford W. Long on the one hundredth anniversary of his discovery of ether as an anesthetic. This is extremely interesting, since a Boston dentist and a Boston physician, Dr. Wells and Dr. Morton, made the discovery at about the same time independently of Dr. Long.

MORRISON, Savannah, Ga.

EFFECT OF TREATMENT WITH TESTOSTERONE PROPIONATE ON MERCURIC CHLORIDE POISONING IN RATS. L. P. LONGLEY, J. Pharmacol. & Exper. Therap. **74**:61 (Jan.) 1942.

Increase in weight of the kidneys, seemingly due to true hypertrophy, has followed injections of androgens in certain laboratory animals, including rats. Mice pretreated with testosterone have been shown to be less susceptible to mercury bichloride poisoning than controls. Longley found that treatment with testosterone propionate of male albino rats poisoned with varying doses of mercury bichloride increased the survival rate of animals which had been given small doses of the mercury compound but was without effect when larger doses of poison were used.

PILCHER, Cleveland.

ACRODERMATITIS AND IDIOPATHIC STEATORRHEA. NIELS DANBOLT, Nord. med. **10**:1390 (May 3) 1941.

Danbolt reports on 2 hypotrophic girls aged 3 years who had suffered from dermatitis since their first year of life and later displayed symptoms of intermittent fat diarrhea. The dermatitis was localized on the face at the eyes, nose and mouth and on the hands, feet, elbows and knees and was resistant to all therapeutic measures. In both cases there were in addition total alopecia and blepharitis. The intestinal disease did not correspond to that of celiac disease. A test showed normal tolerance for dextrose. One of the children had had a sister who died with an illness similar to the one here described. The author is of the opinion that it is a question of a disease entity, and cites some previously reported similar cases.

WALLGREN, Göteborg, Sweden.

# Book Reviews

**Healthy Babies Are Happy Babies.** By Josephine Hemenway Kenyon, M.D. Third edition, revised. Price, $1.50. Pp. 343, with no illustrations. Boston: Little, Brown & Company (Atlantic Monthly Press Book), 1943.

Dr. Kenyon's third revision of her well known book, "Healthy Babies Are Happy Babies," is one of the most complete volumes of its kind.

The first chapter of the book is devoted to good advice for the expectant mother, and the father comes in for a few words of comfort as well. The remainder of the book is concerned with the care of the baby from birth until the age of 3 years, and in each chapter there is additional advice on the care of the mother's health.

Dr. Kenyon has apparently used a wealth of source material, and her advice is in most instances well founded and authentic. One of the chief weaknesses of the book is that the discussion at times becomes too involved and redundant. It is the opinion of this reviewer that instructions to mothers should be as short, clear and concise as they can possibly be made.

In any volume of this type there is, of course, a great deal of room for disagreement among physicians on specific points of infant care. For example, Dr. Kenyon seems to put a great deal of emphasis on "setting-up" exercises for both the mother and the baby. According to the time periods recommended, at least an hour each day would be taken up by these exercises: thirty minutes for the mother and thirty minutes for the baby. It may be questioned whether any normal baby needs to be passively exercised. Babies seem to take about all the exercise necessary if they are not confined too closely by their clothes and bed covering and are given an opportunity to do their own kicking and squirming. Another point with which the reviewer would disagree is the recommended splash of cold water following the bath. This routine has the danger of making the bath time a very unpleasant occasion. It is hard to believe that babies like to have cold water splashed on them at any time. There are many other similar points, some unimportant, some important, with which one might disagree.

Advice given on foods, feeding technic and habit training in general is excellent; one is pleased to note that Dr. Kenyon is careful to avoid rigidity in the recommended routines and gives assurance that allowances must be made for individual variations in different children's reactions to the methods advised, thus getting away from the fault found in so many of the books on child care which create the impression that the same rules can be applied without variation to all individuals.

**The Year Book of Industrial and Orthopedic Surgery, 1942.** Edited by Charles F. Painter, M.D. Price, $3.00. Pp. 424, with 302 illustrations. Chicago: The Year Book Publishers, Inc., 1942.

The annual year book offers a busy practitioner an opportunity to read in reviews the best of the medical publications of the past year. Abstracting the essence, separating the wheat from the chaff requires skilful, experienced editing and determines the value of publications of this kind. The editor, Dr. Painter, is singularly qualified for this task.

He divides the subject matter into chapters covering the major subdivisions of orthopedic surgery. Traumatic surgery is included, since, as Dr. Painter states, there are many good reasons why the two branches should be grouped together. For the first time reviews on the sulfonamide compounds are included. This expresses the editorial policy in presenting reviews only of work that has been shown to have a proved therapeutic value.

Pediatricians will be interested in the articles on the status of tuberculosis of the bones; the early diagnosis of spinal tuberculosis; the revolutionary changes in the management of acute osteomyelitis; curare as a therapeutic agent in spastic paralysis; the notable lack of material on the Kenny treatment of infantile paralysis; chronic arthritis and gold therapy; the introduction of blood and fluids into the general circulation of children by way of the bone marrow, and congenital defects.

There is an excellent series of reviews on fractures and dislocations which foreshadows profound changes in the older standard methods of treatment. The shift toward internal fixation and the influence of war surgery is notable. Special attention is given to painful conditions of the shoulder and to the adjacent scalenus anticus neurocirculatory compression syndrome, which produces a painful arm. About fifty pages of the book contain reviews on

purely industrial medicine, in which the health of workers is involved. Throughout, occasional pertinent editorial comment is found at the close of some of the articles.

This year book is a small, compact volume of 424 pages with over 300 illustrations and is printed in readable type.

**Constitution and Disease.** By Dr. Julius Bauer. Price, $3.50. Pp. 208, with 5 illustrations. New York: Grune & Stratton, Inc., 1942.

Dr. Bauer is well known to many American physicians who attended his lectures on "Endocrinology and Constitutional Pathology" in Vienna, where he was professor of medicine at the university from 1923 through 1938. This book is in part a response to requests for publication of these lectures in book form. The emphasis, however, is on constitution rather than on endocrinology, as the author is of the opinion that the latter field is adequately covered in American literature.

Constitution for the author is synonymous with the genetic "make" of the individual. It is not a definite physical entity but is the "panel and plan of the set up of the individual personality" determined at the time of fertilization of the ovum. It is "a potential energy at work throughout the whole life time determining the physiologic evolutions and involution, the structure and function of the organs, their mutual relation and the response to various stimuli." Recognition of pathologic tendencies due to the presence of abnormal genes is of utmost importance in treating disease and in guiding the life habits of persons so affected.

The subject matter of the book is admirably presented. After a discussion of the significance of constitutional pathology, various phases of abnormalities are considered. First, examples of biologic inferiority of organs and tissues are taken up. From this, one is led to a study of constitutional disturbances of the integrative systems, i. e., the endocrine and nervous systems. Finally, after a consideration of diseases in which the cause is chiefly constitutional, the principles of treatment are elucidated.

This contribution to American medical literature seems especially valuable as the author deals with a phase which has been too little emphasized both in the literature and in the medical schools of this country.

**A Curriculum for Schools of Medical Technology.** Second Edition, Revised. By Israel Davidsohn, M.D., Director of Laboratories and Pathologist, Mount Sinai Hospital, Chicago, and Assistant Professor of Pathology, College of Medicine of the University of Illinois. Price, not given. Pp. 47, with no illustrations. Muncie, Ind.: Registry of Medical Technologists, 1942.

This is a splendid outline for a course for medical technicians and will be of particular value to any one contemplating initiation of a new course. The first chapter is an introduction into medical technology and contains much information valuable to teacher and student alike.

An ideal curriculum is suggested. The subsequent chapters deal with the various divisions of medical technology. Rather than give specific tests, the text refers to a complete and excellent bibliography, so that, in effect, the student has the benefit of the foremost texts on all subjects discussed. The outline is concise yet complete and is well written. It can be highly recommended.

**La glomerulonefritis en la infancia.** By Dr. Arturo Baeza Goñi. Price, not given. Pp. 316, with 14 illustrations. Santiago de Chile: Zig-Zag, 1942.

Written in Spanish, this is an excellent exposition of glomerulonephritis in children. The frequency, etiology, pathology and symptomatology are discussed and illustrated by diagrams, charts and photomicrographs. Considerable space is given to the treatment, both medical and surgical. One feels that the author knows what he is talking about, as 155 case histories are given. The bibliography is complete for both the South American and the foreign literature. This is an interesting book that has an appeal to any physician, regardless of the language he speaks.

**La electroencefalografia.** By Victor Santamarina. Paper. Price, not given. Pp. 170, with 46 figures. Habana: Cardenas y Compania, 1943.

This is an excellent monograph on encephalography, containing the development and experimental work necessary for this phase of diagnosis. There are chapters on the clinical study of electrical cerebral activity in children and on the general clinical applications for all ages. Here especial attention is given to the diagnosis of cerebral lesions, mental states, syphilis of the central nervous system and the use of electroencephalography in aviation medicine. A full and excellent bibliography is included.

# Directory of Pediatric Societies

## INTERNATIONAL

### International Association of Preventive Pediatrics

President: Prof S. Monrad, Dronning Louises Børnehospital, Copenhagen, Denmark.
Secretary: Dr. Daniel Oltramare, 15 Rue Lévrier, Geneva, Switzerland.

### International Congress of Pediatrics

President: Dr. Henry F. Helmholz, Mayo Clinic, Rochester, Minn.
Secretary-Treasurer: Dr. Charles F. McKhann, University Hospital, Ann Arbor, Mich.

Canadian Committee:

Chairman: Dr. Alan Brown, Hospital for Sick Children, 67 College St., Toronto.
Secretary: Dr. H. P. Wright, 1509 Sherbrooke St. W., Montreal.
Place: Boston. Time: Postponed indefinitely.

### International Congress for the Protection of Infancy

Secretary: Prof. G. B. Allaria, Corso Bramante 29, Torino 120, Italy.

## FOREIGN

### Argentine Pediatric Society of Buenos Aires

President: Dr. Martin Ramón Arana, 1809 Rodriguez Peña, Buenos Aires.
General Secretary: Dr. Alfredo Larguia, Cerrito 1179, Buenos Aires.

### British Paediatric Association

President: Prof. L. G. Parsons, 58 Calthorpe Rd., Five Ways, Birmingham, England.
Secretary: Dr. Donald Paterson, 27 Devonshire Pl., London, W. 1.

### Danish Pediatric Society

President: Dr. E. Lenstrup, Copenhagen.
Secretary: Dr. E. Gjørup, Dronning Louises Børnehospital, Copenhagen.

### Nederlandische Vereeniging voor Kindergeneeskunde

President: Dr. J. H. G. Carstens, Servaasbolwerk 14ª, Utrecht.
Secretary: Dr. R. P. van de Kasteele, Laan van Poot 340,'s Gravenhage.
Place: Different places. Time: Three times a year.

### Paediatrický Spolok na Slovensku

President: Dr. A. J. C. Chura, Lazaretská 11, Bratislava.
Secretary: Dr. P. Rados, Lazaretská 6, Bratislava.
Place: Pediatric Clinic, University Bratislava. Time: Six times a year.

### Royal Society of Medicine, Section for the Study of Disease in Children

President: Dr. E. A. Cockayne, 98 Harley St., London, W. 1, England.
Secretary: Dr. R. Lightwood, 86 Brook St., London, W. 1, England.
Place: 1 Wimpole St., London. Time: Fourth Friday of each month, 4:15 p. m.

### Palestine Jewish Medical Association, Section of Physicians of Children's Diseases

President: Prof. S. Rosenbaum, 26 Bialkstr., Tel Aviv.
Secretary: Dr. A. Brünn, 9 Maazestre, Tel Aviv.

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*Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

### Sociedad Cubana de Pediatria

President: Dr. Angel A. Aballí Arellano, 17 No. 609 Vedado, Habana.
Secretary: Dr. Julio G. Cabrera Calderin, Hospital Mercedes L y 21 (Vedado), Box 2430, Habana.
Place: Cátedra de Clinica Infantil, Hospital Mercedes, Habana. Time: Last Wednesday of every month.

### Sociedad Mexicana de Pediatria

President: Dr. Fernando López Clares, 12/a. Medellín 191, Mexico City.
Secretary: Dr. Jesus Gómez Pagola, Versalles 64, Mexico City.

### Sociedad Venezolana de Puericultura y Pediatria

President: Dr. E. Santos Mendoza, Venezuela.
Secretary: Dr. P. Oropeza, Hospital de Niños, Caracas, Venezuela.

### Société de Pédiatrie de Paris

President: Dr. B. Weill-Hallé, 49 Avenue Raymond Poincare, Paris, France.
Secretary: Dr. Jean Hallé, 10 bis Rue Pré aux Clercs, Paris, France.
Place: Hôpital des Enfants Malades, 49 Rue de Sèvres. Time: 4:30 p. m.
Place: Hôpital des Enfants Malades, 49 Rue de Sèvres. Time: 4:30 p. m., third Thursday of every month.

### Uruguayan Society of Pediatrics

President: Dr. Nicolas Leone Bloise, Avenida Agraciada 2496, Montevideo.
Secretary: Dr. Héctor Mourigan, Avenida Agraciada 1464, Montevideo.

## NATIONAL

### American Medical Association, Scientific Assembly, Section on Pediatrics

Chairman: Dr. Hugh L. Dwyer, 315 Alameda Rd., Kansas City, Mo.
Secretary: Dr. Gilbert J. Levy, 188 S. Bellevue Blvd., Memphis, Tenn.

### American Academy of Pediatrics

President: Dr. B. S. Veeder, 3720 Washington Blvd., St. Louis, Mo.
Secretary: Dr. Clifford G. Grulee, 636 Church St., Evanston, Ill.
Region IV. Meeting cancelled.

### American Hospital Association, Children's Hospital Section

Chairman: Dr. Joelle C. Hiebert, 299 Main St., Lewiston, Maine.
Secretary: Dr. W. Franklin Wood, McLean Hospital, Waverly, Mass.
Place: Buffalo, N. Y. Time: Sept. 13-18, 1943.

### American Pediatric Society

President: Dr. James L. Gamble, 300 Longwood Ave., Boston, Mass.
Secretary-Treasurer: Dr. Hugh McCulloch, 325 N. Euclid Ave., St. Louis.

### Canadian Society for the Study of Diseases of Children

President: Dr. R. R. Struthers, 906 Drummond Medical Bldg., Montreal.
Secretary-Treasurer: Dr. Elizabeth Chant Robertson, Hospital for Sick Children, Toronto.

### Society for Pediatric Research

President: Dr. Joseph A. Johnston, Henry Ford Hospital, Detroit.
Secretary: Dr. Mitchell I. Rubin, 1740 Bainbridge St., Philadelphia.

## SECTIONAL

### Intermountain Pediatric Society

President: Dr. Eugene Smith, 385-24th St., Ogden, Utah.
Secretary-Treasurer: Dr. W. C. Cheney, 837 Boston Bldg., Salt Lake City.
Place: Salt Lake City General Hospital. Time: First Thursday of each month, 8 p. m.

NEW ENGLAND PEDIATRIC SOCIETY

President: Dr. Warren R. Sisson, 319 Longwood Ave., Boston.
Secretary-Treasurer: Dr. James Marvin Baty, 1101 Beacon St., Brookline, Mass.
Place: Boston Medical Library. Time: Four meetings a year, occurring from September to May.

NORTH PACIFIC PEDIATRIC SOCIETY

President: Dr. M. L. Bridgeman, 1020 S. W. Taylor St., Portland, Ore.
Secretary: Dr. C. G. Ashley, 833 S. W. 11th Ave., Portland, Ore.

ROCKY MOUNTAIN PEDIATRIC SOCIETY

President: Dr. G. R. Fisher, 23 E. Pikes Peak Ave., Colorado Springs, Colo.
Secretary: Dr. Joseph H. Lyday, 1850 Gilpin St., Denver.

SOUTHERN MEDICAL ASSOCIATION, SECTION OF PEDIATRICS

Chairman: Dr. William Weston Jr., 1428 Lady St., Columbia, S. C.
Secretary: Dr. Angus McBryde, 604 W. Chapel Hill St., Durham, N. C.
Place: Cincinnati, Ohio. Time: Nov. 16-18, 1943.

## STATE

ALABAMA PEDIATRIC SOCIETY

President: Dr. Amos Gipson, 948 Forrest Ave., Gadsen.
Secretary-Treasurer: Dr. Ruth Berrey, 2021–6th Ave. N., Birmingham.

ARIZONA PEDIATRIC SOCIETY

President: Dr. Vivian Tappan, San Clemente, Tucson.
Secretary: Dr. Hilda Kroeger, Arizona State Health Dept. (Maternal and Child Welfare Division), Phoenix.

ARKANSAS STATE PEDIATRIC ASSOCIATION

Chairman: Dr. C. B. Billingsley, 1425 N. 11th St., Fort Smith.
Secretary: Dr. R. E. Weddington, 1425 N. 11th St., Fort Smith.

CALIFORNIA STATE MEDICAL SOCIETY, SECTION ON PEDIATRICS

Chairman: Dr. William C. Deamer, University of California Hospital, San Francisco.
Secretary: Dr. Charles W. Leach, 2000 Van Ness Ave., San Francisco.

FLORIDA STATE PEDIATRIC SOCIETY

President: Dr. Ludo Von Meysenbug, Box 3356, Daytona Beach.
Secretary: Dr. Robert Blessing, 409 Blount Bldg., Ft. Lauderdale.
Place: Concurrent with state association meeting at time of convention.

GEORGIA PEDIATRIC SOCIETY

President: Dr. T. F. Davenport, 104 Ponce de Leon Ave. N. E., Atlanta.
Secretary-Treasurer: Dr. Don F. Cathcart, 478 Peachtree St. N. E., Atlanta.

HEZEKIAH BEARDSLEY PEDIATRIC CLUB OF CONNECTICUT

President: Dr. Edward T. Wakeman, 129 Whitney Ave., New Haven.
Secretary: Dr. Herman Yannet, Southbury Training School, Southbury.
Time: Three meetings a year.

ILLINOIS STATE MEDICAL SOCIETY, SECTION ON PEDIATRICS

Chairman: Dr. Craig D. Butler, 715 Lake St., Oak Park.
Secretary: Dr. A. J. Fletcher, 139 N. Vermilion, Danville.

INDIANA STATE PEDIATRIC SOCIETY

President: Dr. K. T. Knode, 1105 E. Jefferson Bldg., South Bend.
Secretary-Treasurer: Dr. Mathew Winters, 621 Hume Mansur Bldg., Indianapolis.
Time: Two meetings a year.

### Iowa Pediatric Society

President: Dr. Mark L. Floyd, Children's Hospital, Iowa City.
Secretary-Treasurer: Dr. James Dunn, Davenport Bank Bldg., Davenport.

### Medical Society of State of New York, Section on Pediatrics

Chairman: Dr. A. Clement Silverman, 608 E. Genesee St., Syracuse.
Secretary: Dr. Albert G. Davis, 307 Gas and Electric Bldg., Utica.

### Medical Society of the State of Pennsylvania, Pediatric Section

Chairman: Dr. Elwood W. Stitzel, 403 Central Trust Bldg., Altoona, Pa.
Secretary: Dr. P. F. Lucchesi, Philadelphia Hospital, Philadelphia.
Place: Philadelphia. Time: Oct. 5-7, 1943.

### Michigan State Medical Society, Pediatric Section

Chairman: Dr. Charles F. McKhann, University Hospital, Ann Arbor.
Secretary: Dr. Mark F. Osterlin, Central Michigan Children's Clinic, Traverse City.

### Mississippi State Pediatric Society

President: Dr. Harvey F. Garrison Jr., 315 E. Capitol Pl., Jackson.
Secretary: Dr. Guy Verner, 126 N. Congress St., Jackson.

### Nebraska Pediatric Society

President: Dr. Elmer W. Bantin, 440 Aguila Court Bldg., Omaha.
Secretary-Treasurer: Dr. John M. Thomas, 1102 Medical Arts Bldg., Omaha.
Place: As announced by committee. Time: Third Thursday of each month from October to June, inclusive. Dinner at 6 p. m.

### New Hampshire Pediatric Society

President: Dr. MacLean J. Gill, 14 N. State St., Concord.
Secretary-Treasurer: Dr. Ursula G. Sanders, 46 Pleasant St., Concord.
Time: Twice yearly.

### North Carolina Pediatric Society

President: Dr. Thomas M. Watson, Greenville.
Secretary: Dr. Frederick B. Haar, Greenville.

### Oklahoma State Pediatric Society

President: Dr. Ben H. Nicholson, 301 N. W. 12th St., Oklahoma City.
Secretary: Dr. Luvern Hays, 108 W. 6th St., Tulsa.
Place: Oklahoma Club. Time: 6:30 p. m., fourth Friday of each alternate month from September to May, inclusive.

### South Carolina Pediatric Society

President: Dr. Lonita Boggs, 301 E. Coffee St., Greenville.
Secretary-Treasurer: Dr. Hilla Sheriff, Wade Hampton Office Bldg., Columbia.

### Texas Pediatric Society

President: Dr. F. H. Lancaster, 4409 Fannin St., Houston.
Secretary-Treasurer: Dr. John Ashby, 3610 Fairmount Ave., Dallas.

### Virginia Pediatric Society

President: Dr. J. S. Weitzel, 3103 W. Franklin St., Richmond.
Secretary: Dr. Emily Gardner, 1100 W. Franklin St., Richmond.

### West Virginia State Medical Society, Section on Pediatrics

President: Dr. Andrew Amick, 1021 Quarrier St., Charleston.
Secretary: Dr. A. A. Shawkey, Professional Bldg., Charleston.

## LOCAL

### Academy of Medicine of Cleveland, Pediatric Section

Chairman: Dr. J. D. Nourse, 10515 Carnegie Ave., Cleveland.
Secretary: Dr. I. B. Silber, 10465 Carnegie Ave., Cleveland.
Place: Cleveland Medical Library Bldg. Time: October, December, February and April.

### Academy of Medicine, Toronto, Section of Pediatrics

President: Dr. I. Nelles Silverthorne, 170 St. George St., Toronto, Canada.
Secretary: Dr. G. P. Hamblin, 2333 Bloor St. W., Toronto, Canada.

### Bronx Pediatric Society

President: Dr. Forris E. Chick, 2325 University Ave., New York.
Secretary: Dr. Jacob H. Turner, 121 W. 197th St., New York.
Place: Concourse Plaza Hotel, 161st St. and Grand Concourse. Time: Every second Wednesday of each month, except June, July, August and September.

### Brooklyn Academy of Pediatrics

President: Dr. Charles A. Weymuller, 85 Pierrepont St., Brooklyn.
Secretary: Dr. Lewis A. Koch, 62 Pierrepont St., Brooklyn.
Place: Granada Hotel. Time: Fourth Wednesday of October, November, February, March and April.

### Buffalo Pediatric Society

President: Dr. A. Wilmot Jacobsen, 187 Bryant St., Buffalo, N. Y.
Secretary: Dr. Richard A. Downey, 786 Forest Ave., Buffalo, N. Y.
Place: Children's Hospital, 219 Bryant St. Time: 8:30 p. m., first Monday of each month from September to June.

### Central New York Pediatric Club

President: Dr. Edward J. Wynkoop, 501 James St., Syracuse.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany.
Places: Various cities in New York. Time: Third Tuesday of April and September.

### Chicago Pediatric Society

President: Dr. Heyworth N. Sanford, 952 N. Michigan Ave., Chicago.
Secretary: Dr. Henry G. Poncher, 1819 W. Polk St., Chicago.
Place: Children's Memorial Hospital, 710 Fullerton Ave. Time: Third Tuesday of each month, October to May, inclusive.

### Cincinnati Pediatric Society

President: Dr. Lloyd K. Felter, 3144 Jefferson Ave., Cincinnati.
Secretary: Dr. T. Selkirk, 3530 Reading Rd., Cincinnati.
Place: Children's Hospital, Elland Ave., Cincinnati. Time: On call.

### Dallas Pediatric Society

President: Dr. John E. Ashby, 3610 Fairmount Ave., Dallas, Texas.
Secretary-Treasurer: Dr. Gladys J. Fashena, 4585 Belfort, Dallas, Texas.
Place: Bradford Baby Hospital. Time: 1 p. m., second and fourth Saturdays of each month.

### Detroit Pediatric Society

President: Dr. Hugh Lewis, Detroit, Mich.
Secretary: Dr. John J. Pollack, 622-26 Maccabees Bldg., Detroit, Mich.
Place: Wayne County Medical Society. Time: 8:30 p. m., first Wednesday of each month from October to June, inclusive.

### Fulton County Medical Society, Pediatrics Section (Atlanta, Ga.)

Chairman: Dr. Don F. Cathcart, 478 Peachtree St. N. E., Atlanta.
Secretary: Dr. Harry Lange, 478 Peachtree St., N. E., Atlanta.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: Second Thursday of each month from October to April, 8 p. m.

### Houston Pediatric Society

President: Dr. Raymond Cohen, 2300 Caroline St., Houston, Texas.
Secretary: Dr. Betty Moody, 526 Richmond Rd., Houston, Texas.
Place: College Inn, Houston. Time: Fourth Monday of each month.

### Kansas City (Missouri) Pediatric Society

President: Dr. Edwin H. Schorer, 1103 Grand Ave., Kansas City.
Secretary: Dr. H. E. Petersen, Kirkpatrick Bldg., St. Joseph, Mo.
Place: Kansas City General Hospital. Time: On call.

### Los Angeles County Medical Association, Pediatric Section

President: Dr. Oscar Reiss, 2200 W. 3d St., Los Angeles.
Secretary-Treasurer: Dr. Elena Boder, 1830½ Lucille Ave., Los Angeles.
Place: Los Angeles County Medical Headquarters, 1925 Wilshire Blvd. Time: Second Monday of February, April, June, October and December.

### Medical Society of the County of Kings and the Academy of Medicine of Brooklyn, Pediatric Section

President: Dr. Harry Bikoff, 850 St. Marks Ave., Brooklyn.
Secretary: Dr. Henry Rascoff, 895 Eastern Parkway, Brooklyn.
Place: 1313 Bedford Ave., Brooklyn. Time: 9:00 p. m., fourth Monday of each month, October to April, inclusive.

### Medical Society of the County of Queens, Inc., Section on Pediatrics

Chairman: Dr. Meyerón Coe, 217-02-91st Ave., Queens Village, N. Y.
Secretary-Treasurer: Dr. Edith A. Mittell, 144-38th Ave., Flushing, N. Y.
Place: Queens County Medical Bldg., Forest Hills, N. Y. Time: Third Monday of October, January, March and May.

### Medical Society of the District of Columbia, Section on Pediatrics

President: Dr. Harry A. Spigel, 2647 Connecticut Ave., Washington, D. C.
Secretary-Treasurer: Dr. Perry W. Gard, 2520 Woodley Rd., Washington, D. C.
Place: Medical Society Bldg., 1718 M St. N. W. Time: 8 p. m., fourth Thursday of every month.

### Memphis Pediatric Society

President: Dr. F. T. Mitchell, 376 S. Bellevue Ave., Memphis, Tenn.
Secretary-Treasurer: Dr. Harry Jacobson, 1193 Madison Ave., Memphis, Tenn.
Place: John Gaston Hospital. Time: Quarterly.

### Milwaukee Pediatric Society

President: Dr. John H. Reynolds, 1628 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. F. J. Mellencamp, 324 E. Wisconsin Ave., Milwaukee.
Place: Milwaukee Athletic Club. Time: Second Wednesday of each alternate month, beginning with February.

### New York Academy of Medicine, Section of Pediatrics

Chairman: Dr. Edith M. Lincoln, 600 Park Ave., New York.
Secretary: Dr. John D. Lyttle, Babies Hospital, New York.
Place: New York Academy of Medicine, 2 E. 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

### Northern California Affiliates

President: Dr. Crawford Bost, 490 Post St., San Francisco.
Secretary: Dr. William A. Reilly, 384 Post St., San Francisco.
Time: Second Thursday of September, November, January, March and May.

### Northwestern Pediatric Society

President: Dr. Arild E. Hansen, University of Minnesota, Minneapolis.
Secretary-Treasurer: Dr. Albert V. Stoesser, 205 W. University Hospital, Minneapolis.
Place: Minneapolis, St. Paul, Duluth and Rochester. Time: January, April, July and October.

### Oklahoma City Pediatric Society

President: Dr. William M. Taylor, Oklahoma City.
Secretary: Dr. G. R. Felts, 625 N. W. 10th St., Oklahoma City.
Place: Oklahoma Club. Time: Third Thursday of each month.

### Philadelphia Pediatric Society

President: Dr. Theodore S. Wilder, Greene and Coulter Sts., Germantown, Philadelphia.
Secretary: Dr. Sherman Little, 1740 Bainbridge St., Philadelphia.
Place: College of Physicians, 19 S. 22d St. Time: Second Tuesday in March, May, October and December.

### Pittsburgh Pediatric Society

President: Dr. Carl Ruder, 646 Washington Rd., Mount Lebanon, Pittsburgh.
Secretary-Treasurer: Dr. C. J. Stoecklein, 125 De Soto St., Pittsburgh.
Place: Pittsburgh Academy of Medicine. Time: Second Friday, alternate month from October to June, inclusive.

### Richmond Pediatric Society

President: Dr. Stanley Meade, 913 Floyd Ave., Richmond, Va.
Secretary-Treasurer: Dr. Louise Galvin, 214 S. Boulevard, Richmond, Va.
Place: Richmond Academy of Medicine, 1200 E. Clay St. Time: 8 p. m., third Thursday of each month, except June, July and August.

### Rochester Pediatric Society

President: Dr. Herbert Soule, 122 Rutgers St., Rochester, N. Y.
Secretary-Treasurer: Dr. Jerome Glaser, 300 S. Goodman St., Rochester, N. Y.
Place: Rochester Academy of Medicine or arrangement by program committee. Time: Third Friday of each month from October to May.

### St. Louis Pediatric Society

President: Dr. Jerome Diamond, 508 N. Grand Ave., St. Louis.
Secretary-Treasurer: Dr. Mary A. McLoon, 408 Humboldt Bldg., St. Louis.
Place: St. Louis Medical Society Bldg. Time: First Friday of each month from November to June.

### Seattle Pediatric Society

President: Dr. Frederick B. Joy, Stimson Bldg., Seattle.
Secretary: Dr. Sherod M. Billington, Medical Dental Bldg., Seattle.
Place: College Club. Time: Third Friday of each month from September to June at 6:30 p. m.

### Southwestern Pediatric Society

President: Dr. Jeanette Harrison, 1136 W. 6th St., Los Angeles.
Secretary: Dr. Henry F. Gallagher, 1930 Wilshire Blvd., Los Angeles.
Place: Jonathan Club of Los Angeles. Time: First Wednesday in January, March May, September and November.

### University of Michigan Pediatric and Infectious Disease Society

President: Dr. Campbell Harvey, 35 W. Huron St., Pontiac, Mich.
Secretary: Dr. Harry A. Towsley, University of Michigan, Department of Pediatrics and Communicable Diseases, Ann Arbor, Mich.

### Westchester County Medical Society, Pediatrics Section (New York)

President: Dr. John B. Ahouse, 27 Ludlow St., Yonkers, N. Y.
Secretary-Treasurer: Dr. Elvira Ostlund, 64 Highland Rd., Rye, N. Y.
Place: Grasslands Hospital, Valhalla, N. Y. Time: Third Thursday in October, December, February and April.

THE AMERICAN JOURNAL OF DISEASES OF CHILDREN is published by the American Medical Association as a medium for the advancement of knowledge of the diseases of children and for the publication of scientific investigation in this field.

Manuscripts for publication, books for review and correspondence relating to the editorial management should be sent to Dr. Clifford G. Grulee, Chief Editor, 636 Church Street, Evanston, Ill., or to any other member of the Editorial Board. Communications regarding subscriptions, reprints, etc., should be addressed, AMERICAN JOURNAL OF DISEASES OF CHILDREN, American Medical Association, 535 North Dearborn Street, Chicago 10.

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Footnotes and bibliographies (the latter are used only in exhaustive reviews of the literature) should conform to the style of the *Quarterly Cumulative Index Medicus*. This requires, in the order given: name of author, title of article and name of periodical, with volume, page, month—day of month if the journal appears weekly—and year.

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Checks, money orders and drafts should be made payable to the American Medical Association.

## OTHER PERIODICAL PUBLICATIONS
### of the American Medical Association

**THE JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION**—Weekly. Covers all the medical sciences and matters of general medical interest. Illustrated. Annual subscription price (three volumes): domestic, $12.00; Canadian, $13.50; foreign, $15.00. Single copies, 25 cents.

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